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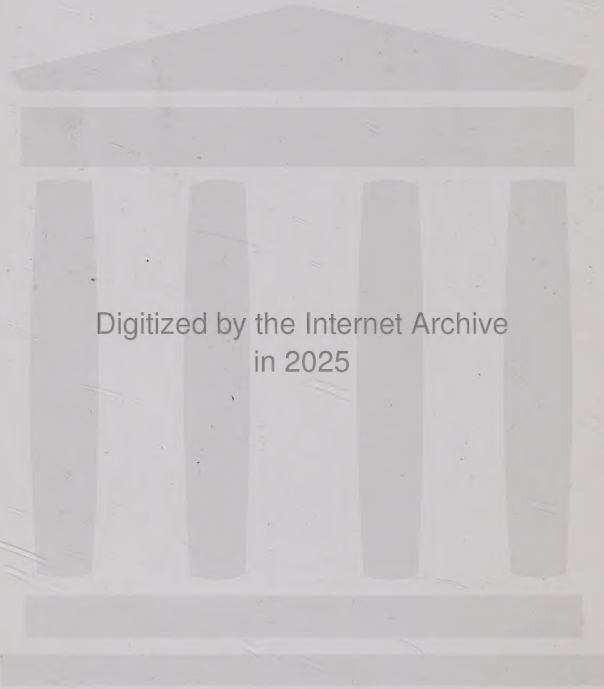
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HANDBOOK
OF
MEDICINE AND THERAPEUTICS

WHEELER AND JACK

HANDBOOK

OF

MEDICINE & THERAPEUTICS

BY

ALEXANDER WHEELER, L.R.C.P. & S.E.

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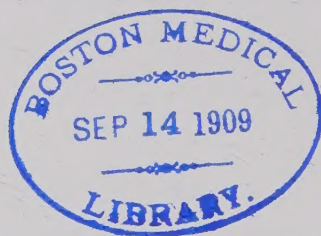
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PREFACE TO THE THIRD EDITION

THE gratifying reception recorded to the last edition of Dr. Wheeler's *Handbook of Medicine and Therapeutics*, which has been exhausted in the comparatively short period of four years, is at the same time an indication that it has, in some measure, accomplished the objects for which it was intended, and a stimulus towards further improvement of the new edition now called for. The chief aims which Dr. Wheeler held before himself (I quote from the Preface to the first edition) were these:—

“1. To enable the Student to digest the main features of the various diseases in the most concise manner at the time he is engaged in clinical work, thereby enabling him to verify *at the bedside* the statements he has read.

“2. To enable the busy practitioner to see at a glance the principal points of each disease, which he can elaborate by his experience.”

But it was more than a mere compendium of symptoms at which Dr. Wheeler aimed. His further object was to protest against the system of learning without understanding, of learning a disease by symptoms without correlating them with the deviations from healthy function which produced them. It was therefore a feature of his work, introduced perhaps for the first time in so small a text-book, that he prefaced his account of the diseases of special organs, wherever it seemed to be necessary, by a brief summary of the anatomy or functions of those organs in health, and that he not only

described the physical signs of disease, but explained their production. These objects, both in the last and in the present edition, I have endeavoured to bear in mind. The very considerable modifications made in the last edition have been further extended in the present. The arrangement of the book has been materially altered, and the order in which the diseases of the different systems have been discussed has been recast in favour of a more natural sequence. Between the first and second editions over nine years elapsed, between the second and the present edition four years only. Evidently, therefore, the progress of knowledge required more radical additions and alterations in the second edition than in the present, but there has been room here also for much improvement. In a handbook of so small a size it is impossible to take note of all the most recent discoveries or the newest theories, but it is hoped that the most important have found due recognition, the aim being to present a brief but clear description of the most important facts of medicine in such a fashion as to form a useful complement to the bedside study of disease.

WILLIAM R. JACK.

43 LANSDOWNE CRESCENT,
GLASGOW, 1908.

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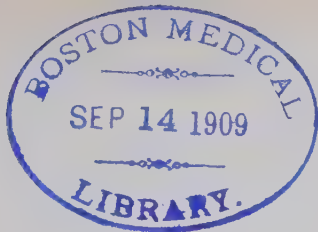
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HANDBOOK

OF

MEDICINE AND THERAPEUTICS



SPECIFIC INFECTIOUS DISEASES

FEVERS.

FEVERS are characterised by a grave disturbance of the system, attended with great *increase of temperature* and *diminished secretions*; or in other words, *more fire* and *greater accumulation of ashes*. All the specific or "zymotic" diseases are probably due to special germs setting up chemical or fermentative changes in the blood. Some of these organisms we know something about, of others we know nothing. Pasteur was the first to elaborate the fermentation theory.

Suppose we put into a bottle—water, sugar, and barm; then cork it tightly, and let it stand in a warm place. At first *nothing* seems to happen, but after a time a remarkable change takes place—CO₂ and alcohol form; and a sediment sinks to the bottom, that is, the *sugar has been changed by the living yeast cells into poisonous compounds*; this process stops, as so many waste and poisonous products are formed that the *yeast cells themselves are killed*!

Again,—put into a bottle some sugar and a solution of *nitrogenous* material (say, clear mutton broth), and instead of corking, *expose it to the air*. Again fermentation takes place, and a scum soon rises to the surface. If the scum be examined under the microscope it will be found to consist of millions of cells = bacteria. The once harmless fluid *has become highly poisonous*; it rapidly decomposes, and gives rise to a most disgusting odour.

Here, then, are two cases of fermentation: the one induced by cells *introduced by human agency*, the other by bacteria *which got in from the air*. In both cases, poisons were formed, and the *bacteria*, like the yeast cells, were killed through their own products, or through exhaustion of suitable food, and thus the fermentative process was stopped.

If this be applied to specific fevers in the young, for instance scarlet fever, it is easily seen that a period must elapse after the settlement of the organisms, and during their development, before definite symptoms are produced. The patient will probably in that stage have symptoms of the kind known as malaise. Later, when either the organisms or the toxins have got into the blood stream, there occurs the febrile and protective reaction, with the specific symptoms of the particular disease. If the reaction is adequate, there follows a decline of symptoms, leading to convalescence. If it is inadequate, there follows an overcoming of the systemic resistance by the toxins, leading to the ultimate stage, still known as the “typhoid state” (for symptoms see p. 5) and finally to death.

Acute infective diseases arising from microbic infection therefore have—

1. A period of incubation.
2. A period in which the organisms multiply, and send forth toxins, leading to the characteristic symptoms of the particular disease.
3. A period, in favourable cases, of decline, where the bodily resistance has overcome the organism. The temperature may fall rapidly, by *crisis*, or more gradually, by *lysis*.

It is not, however, only by the direct action of the organisms that fever is produced. They may be entirely localised to a particular part of the body, but the products of their vitality may enter the circulation, and cause, besides fever, the phenomena of toxæmia, on which many of the nervous and cardiac symptoms largely depend (delirium, cardiac irregularity or tachycardia, and so on). Fever, it may be more than plausibly said, is a reaction of the previously healthy body against the organisms of disease and their products. In many instances the reaction is excessive, and destroys by its over-exuberance the life it was meant to guard. But that it is a protective reaction is shown in the diseases of the old. In them, where the body has no longer its former resistant power, there is little or no fever, even in such affections as pneumonia. A young man, previously healthy, may regain perfect health after a pneumonic attack, although in its duration his temperature may have risen to 105° or more. An old man suffering from pneumonia may have a temperature but little elevated, and yet the disease almost inevitably tends towards death. In the one case the febrile reaction expresses the resistance of the body; in the other its absence expresses a failing power to combat either micro-organisms or their toxins.

The indications for treatment, therefore, are evidently—

First—to prevent the formation of suitable soils by hygienic measures.

Secondly—to make an already suitable soil *unsuitable*, by antiseptics, stimulants, and tonics; in other words, fight the bacteria by making the resistance greater, through increasing the vitality of the tissues.

Thirdly—in certain instances (diphtheria, etc.), by the use of antitoxins, to administer a direct antidote to the toxins formed by the specific micro-organism, or produce immunity in those exposed to the disease.

To sum up: decrease if possible the number of bacteria attacking, and make the soil more resistant; also support the patient's strength till the fight is finished. Let me impress

upon beginners, however, that "prevention" promises a far better prospect than cure: for bacteria, once they flourish vigorously in the body, are very difficult to exterminate.

Morbid Anatomy of Fevers. General Changes.—Each fever has its special foci, but usually in addition to their *peculiar* lesions, *all, when severe*, show more or less change in the tissues generally, viz.—

1. Blood is dark and more fluid (thick in cholera). There is usually an increase in the number of leucocytes, known as leucocytosis.

2. Muscles dark-coloured, and show granular degeneration.

3. Heart is softened, perhaps dilated.

4. Congestion of the viscera, *spleen, liver, and kidneys* particularly.

5. Hypostatic congestion of lungs.

6. In consequence of these changes, diminution in all the secretions, except that of sweat. The skin may be dry, or sweating may be profuse.

PHYSIOLOGY OF FEVER.

Before discussing the treatment of fever we must consider a few physiological questions. In fever we have a disturbance of the heat-regulating centre, so that in addition to the pernicious products of the specific germ, *we have also to consider the excessive waste of tissue, and diminished excretion*, with, consequently, the accumulation within the blood of tissue waste products, which are also poisonous in their nature. Thus a cycle of retrograde changes commences. The liver being engorged does its work less efficiently, proteid waste is not broken down so completely, and the kidney has to excrete *not only a greater amount of waste nitrogenous material, but material of a more irritating nature*, and this too, when by reason of the deterioration of the blood, the organ itself is but poorly nourished. Excessive heat production, along with diminished loss, is maintained so long as the causative toxin continues to

circulate in the blood. It is this disproportion which accounts for the rigor so often occurring at the beginning of fevers, spasm of the cutaneous vessels producing a sense of cold, and giving rise to shivering by stimulation of the sensory nerves. The rigor ceases so soon as the muscular spasm producing it has caused sufficient heat to warm the skin. From similar causes, the apparent heat of the skin forms no true indication of the internal temperature. Thus in ague the skin is at first quite cold, while the rectal temperature is many degrees above normal.

In describing any fever, begin with the *marked* features of the invasion, *then* emphasise—

1. The *special* symptoms of the fever.
2. The manner in which it declines.
3. The complications.
4. The sequelæ.

Remember that *all* fevers have more than one type ; always at least two—the simple and the severe ; some, indeed, have many forms, such as small-pox and scarlet fever.

All may assume what is termed the *Typhoid State*—a condition of great gravity. In order to avoid constant repetition, we shall in the following pages describe only the peculiar features of each “malaise,” and sum up the later and more severe symptoms of each fever under the term “Typhoid State.” It is to be particularly noted that this condition is *not* synonymous with Typhoid Fever.

TYPHOID STATE.

The Typhoid State is characterised by—

1. A decline of the previously more acute symptoms.
2. Pulse becoming rapid and soft.
3. The tongue is dry and brown, tremulous, and protruded with difficulty.
4. Collection of sordes (a mixture of dried mucus and bacteria) around the teeth and lips.

5. Increased muscular prostration—tremors and subsultus tendinum.

6. Semi-comatose state, coma vigil = pupils dilated, but patient does not see.

7. Picking at the bed-clothes and muttering delirium.

8. Slipping down to the foot of the bed.

This condition becomes well marked in *all* the malignant forms of fevers—is very well seen in small-pox, typhus, typhoid, pneumonia, etc.

GENERAL TREATMENT OF ALL FEVERS.

I. Medicinal.—The indications for treatment are sufficiently obvious—

1. Where possible, attack the exciting cause (use of quinine in malaria, etc.).
2. Diminish heat production.
3. Increase heat loss.
4. Help the secretory organs.
5. Keep the heart going.
6. Treat complications as they arise.

Under the second heading we may employ antipyretics—

- Drugs—(1) Antipyrin group.
(2) Salicylates.
(3) Quinine.
(4) Aconite.
(5) Stimulants.

Of these, such as do not depress the heart are to be preferred, and should generally be given only when the fever is prolonged or excessively high. Cold sponging and cold baths are often valuable.

Under the third heading we may choose—

Diaphoretics—Liq. Ammon. Acet.

Alkaline group generally.

To help the secreting organs—

- (1) Diminish the amount of nitrogenous food, and substitute milk and farinaceous material.
- (2) Keep the bowels moderately open. *Make the urates more soluble* by administration of citrates, tartrates, etc.

And lastly, to keep the heart going, we may employ—

- (1) Digitalis.
- (2) Strophanthus.
- (3) Strychnine.
- (4) Diffusible stimulants, brandy, ether.

No routine treatment can be prescribed, because of the many complications and peculiarities of each case. In most cases we may start with a purge, if there be no contra-indication.

℞ Hyd. Subclor. grs. iv. ; followed in 4 hours by
℞ Mist. Sennæ Co., ℥ss.

This may be combined, in mild cases, with a mixture such as the following, taken at intervals, say, a tablespoonful every four hours—

℞ Liq. Ammon. Acet. . . ℥ij.
Spt. Ætheris Nitrosi . . ℥ss.
Potass. Citratis . . . gr. xx.
Aquæ Camph. ad . . ℥ss.

This will keep skin and kidneys acting, the citrate also rendering the urates less irritating.

Slight degrees of fever do not, however, require active treatment, as they are simply the expression of the protective reaction of the body against the cause of disease.

II. Hygiene.—Frequently “*drugs*” will form the least important factor in successful treatment, and in *all* cases their action will be assisted by careful diet and strict attention to hygienic principles.

The essentials are—

1. A large well-ventilated room, with blinds which may be

so arranged as to let in plenty of light when wanted, or to exclude light if necessary.

2. An absence of unnecessary furniture, which only serves to form a nidus for retention of the germs. Carpets and curtains are particularly objectionable, and should be removed in any serious fever.

3. A well-trained, non-officious nurse.

4. Absolute cleanliness.

5. Strict attention to physician's orders.

6. Disinfection of all excreta.

ALCOHOL IN DISEASE.

It is undoubtedly the case that the great diminution in the use of alcohol in disease, which has characterised recent practice in every country, has been attended by no increase in mortality; rather the reverse. The old routine administration of alcohol in fevers cannot be too strongly condemned. Many cases recover perfectly without a drop of alcohol from onset to convalescence. But the rigorous prohibition of it in every case would seem to be almost as unreasonable. In those who have for long been accustomed to its use in health, its sudden withdrawal in illness may lead to anorexia, and hence to lowered vitality. There are, too, conditions of emergency in which the temporary use of alcohol may tide over a pressing danger. The individual symptoms must be studied in every case, and to-day, as when it was written, Brunton's summary applies:—If the alcohol tends to bring the patient nearer his normal condition it is doing good; if it takes him away farther from his healthy condition, it is doing harm. For instance, if alcohol renders the tongue moist, slows the *quicken*ed pulse or the hurried breathing, if it renders the skin cooler when hot and dry, and if it lessens delirium and brings on sleep, then use it. If the converse happens, then withhold it—typhoid or any other condition.

Alcohol is chiefly indicated during the small hours of the

morning, when attendants are sleepy ; the fire perhaps gets low and the external temperature is generally lowered. (WHITLA.)

DELIRIUM

Is such a common complication, we may take it here. There are three types commonly met with—

1. Raving maniacal delirium, usually associated with great violence and extreme muscular activity.

2. Low muttering delirium of later stages, in which the patient lies still and sunk in bed, and babbles incoherently.

3. Delirium tremens may be met with in any fever complicated by alcoholism, or apart altogether from the fevers. It is characterised in particular by restlessness, tremor, and hallucinations of sight and hearing.

In the first case we may employ sedatives or depressants, as bromide of potassium, either alone or with chloral hydrate, opium, or antipyretics to control the fever which causes the delirium. Ice to the shaved head, and cold sponging are also useful.

The following formula may prove useful, where the heart is not already weakened—

R	Chloral Hydrat.	℥ss.
	Potass. Brom.	gr. xl.
	Aq. ad	℥ij.

One half at once, the other half repeated in one hour if required.

The second type must be met by stimulants, as carbonate of ammonia or alcohol in full doses either by the mouth or in the form of enema. If the temperature is high, such antipyretics as quinine in full doses should be used. We may indeed sum up in similar words the *treatment of the typhoid state*.

Delirium tremens is to be treated by careful nursing, frequent giving of fluid food, and the use of hypnotics and sedatives. Bromide, or bromide and chloral, or paraldehyde in large doses may be used, and if they fail, hyoscine injections, beginning with not more than $\frac{1}{200}$ gr.

INCUBATION AND RASHES.

The period of incubation in fevers is the time intervening between the reception of the specific virus and the onset of symptoms.

The *average* duration of the period is here given for the more important diseases—

Cholera	4 to 5 days.
Diphtheria	2 days.
Enteric fever	10 to 14 days.
Influenza	3 days.
Measles	14 days.
Plague	3 to 5 days.
Scarlatina	2 days.
Typhus fever	12 days.
Varicella (chicken-pox)	14 days.
Variola (small-pox)	12 days.

(DAWSON WILLIAMS.)

The period of incubation must be distinguished from the period of onset of the characteristic rash, which may occur along with the first symptoms, or some time later.

The following table is fairly correct. The rash appears in—

Chicken-pox	1st day <i>of the disease.</i>
Scarlet Fever	1st or 2nd day „
Small-pox	3rd day „
Measles	4th day „
Typhus	5th day „
Enteric	7th to 12th day „

Most rashes disappear on pressure, *if not hæmorrhagic*. If hæmorrhagic they do not disappear on pressure *or after death*.

ENTERIC or TYPHOID FEVER.

A specific infectious fever marked clinically by diarrhœa and a rose-coloured rash (appearing in successive crops), running a prolonged course of about twenty-one days, and ending by lysis. It is accompanied by characteristic ulcerations of the small intestine.

Ætiology.—Prevails most in autumn months. Attacks both sexes from fifteen to twenty-five years of age. Rare in infancy or over the age of sixty.

The poison is conveyed principally by—

1. Water contaminated by stools.
2. Soiled linen of typhoid patients in public laundries.
3. Milk.
4. Drains.
5. Direct contact with stools of patients.
6. Contamination of the soil. (PETTENKOFER.)
7. Ingestion of shell-fish, especially oysters, which have been grown upon beds in the estuaries of rivers the water of which is polluted by sewage.

The possibility of air-borne typhoid must be remembered. The organism may be drawn into the mouth in dust, and afterwards swallowed.

The stools when *fresh* are said to be less poisonous than after fermentation has commenced, the most virulent period being from twelve to thirty-six hours after having been passed.

Sewer gas, filth, etc., do not of themselves cause typhoid fever, but cause a suitable soil for the typhoid bacillus to multiply in the intestines.

Specific Germ—known as the bacillus of Eberth. It is short, thick, and has rounded ends. It is highly mobile, and possesses numerous flagella. It does not form spores. Cultures are easily destroyed by corrosive sublimate 1-2500. During life the bacilli may be found in the stools and urine. They are discoverable only with difficulty in the peripheral blood. *Post mortem* they are found in the intestinal and mesenteric glands, the spleen, the blood, and sometimes in other parts of the body.

Morbid Anatomy.—Principally inflammation of the lymphoid tissue of the lower portion of the ileum, with more or less catarrh throughout the bowel.

Peyer's Patches—

1st Week.—Are swollen through infiltration of leucocytes, the surfaces raised and fawn-coloured—the infiltration involves the submucous coat. The lesions are most numerous at the lower end of the ileum.

2nd Week.—The surface becomes abraded; sloughs form, which are often bile-stained.

3rd Week.—Sloughs come away, leaving ulcerating surfaces. Typical typhoid ulcers are thus formed. A few solitary glands undergo the same process. At the end of the week the ulcers begin to granulate, but healing is usually slow.

Mesenteric Glands may undergo the same changes, but more often become swollen, red, and tender only, or break down into cheesy masses.

Other Organs.—Spleen and liver are enlarged; heart is soft and flabby. The voluntary muscles undergo granular degeneration; in fact, similar changes to those found after death from high temperature, etc. (See page 4.)

Symptoms.—*Insidious onset*—headache, epistaxis, increasing weakness till patient is compelled to take to bed.

1st Week.—Temperature runs up in remarkable staircase manner—*i.e.*, rising two degrees in the evening and falling *one* in the morning. (Not always.) The malaise becomes more marked, and exhaustion increases with the onset of—

- (1) Diarrhœa—the stools quickly assuming their characteristic appearance. The bowels may, however, be confined throughout.
- (2) Tenderness in the right iliac fossa, tumidity of the abdomen, and enlarged spleen. This enlargement is more obvious at a later stage.

The specific rash appears on the seventh to twelfth day, and may continue throughout the disease.

2nd Week is marked by the symptoms becoming more aggravated; the temperature remains at a uniform level. A tendency to *typhoid state* comes on, rambling delirium may occur towards the end of the week, and death may take place.

3rd Week is marked by even more profound symptoms.

The heart is weak and rapid, there is hypostatic congestion of the lungs, the abdomen is distended, and the typhoid state is marked. Perforation is much more likely to occur at this than any other period, or perhaps fatal epistaxis or the pneumonic condition may end the scene.

4th Week.—The temperature gradually becomes normal, and usually convalescence may be now said to *commence*. Frequently, however, relapses occur.

Complications of typhoid fever are—

1. Hæmorrhage; from bowel, nose, or mucous surfaces.
2. Perforation, which may occur even in mild cases. It is commonest in the third week.
3. Peritonitis, with or without perforation.
4. Thrombosis, usually of the femoral vein, and embolism.
5. Meningitis.
6. Pneumonia, lobar in the early stages, later catarrhal.
7. Early "typhoid state," or early cardiac failure.
8. Hyperpyrexia.

SPECIAL POINTS.

The *Rash* comes out in successive crops, on the seventh to twelfth day, as small rose-coloured spots raised above the surface, slightly convex; they appear first on the abdomen and chest; and fade on pressure. Each spot lasts about four days, and the eruption may persist till the end of the fever. Frequently no rash appears (in about 30 per cent of the cases).

The *Ulcer* of Typhoid fever—

Lies in the longitudinal axis.

Edges—thin, undermined.

Situation—last yard of ileum; ulcers most numerous near ileo-colic valve. The distribution may be more wide-spread.

Base—may be formed of—

1. Sub-mucous coat.
2. Muscular tissue, or
3. Peritoneum only.

The ulcers show a tendency to perforate, but *do not cause constriction after they heal.*

The Stools are liquid, abundant, and fœtid; like pea soup in appearance, and contain, besides the bacillus, fæcal matter, sodium chloride, triple phosphates, and frequently blood, shreds of mucous membranes, and sloughs. When hæmorrhage occurs, they become black or tarry in appearance, and sticky. In severe hæmorrhage the temperature falls.

Leucocytosis is not a feature of uncomplicated enteric fever. When it does occur, it indicates some inflammatory complication.

	TYPHUS.	TYPHOID.
<i>Age</i> . . .	Males, 25 to 35.	Both sexes, 15 to 25.
<i>Cause</i> . . .	Dirt; filth; overcrowding.	Contamination with typhoid stools; defective drainage.
<i>Onset</i> . . .	Abrupt.	Insidious.
<i>Course</i> . . .	Ends by crisis on fourteenth day.	By lysis after the twenty-first day; frequent relapses.
<i>Chief Symptoms</i> .	Referable to the nervous system; more delirium, and early appearance of typhoid state.	Referable more to the alimentary canal; nervous symptoms and typhoid state later.
<i>Pupils</i> . . .	Contracted.	Dilated.
<i>Bowels</i> . . .	Confined.	Loose.
<i>Rash</i> . . .	Dark dusky mulberry; comes out on fifth day; tends to become hæmorrhagic.	Rose-coloured, like flea-bites; appears seventh to twelfth day in successive crops.
<i>Temperature</i> .	Goes up rapidly, and comes down suddenly.	Rises 2° in the evening; falls 1° in the morning for four days; then oscillates throughout the disease.

Signs of perforation.—Sometimes there is pain, sometimes none. There are rapid and extreme distension of abdomen, absence of abdominal respiration, tympanitic percussion note, absence of hepatic and splenic dulness. There are also signs of *shock*, anxious look, very rapid pulse, rapid respiration.

Widal's reaction.—Serum from a case of enteric fever will, when mixed with an emulsion of *B. typhosus*, produce agglutination of the bacilli into clumps, even when the serum is diluted to 1 in 20-50 with sterile bouillon. To carry out the test, blood is collected from a puncture in the skin by a small pipette, in which it is allowed to coagulate, and the serum then blown out and diluted to the desired degree (1 in 20 or 1 in 50). Equal parts of this and the emulsion are then mixed together, and the result watched under the microscope in a hanging-drop preparation, or in a small glass tube sealed at one end, and set vertically. If the case be one of enteric, agglutination should take place within from half an hour to an hour. The test does not give positive results, as a rule, until about the seventh or eighth day.

The Diazo-reaction of Ehrlich.—To forty parts of a saturated solution of sulphanilic acid in 5 per cent hydrochloric acid add one part of a 0.5 per cent solution of sodium nitrite. Shake up a drachm of this with a drachm of the patient's urine in a test tube, and float upon the surface a layer of liquor ammoniæ. A ring of red colouration is formed at the point of contact. On shaking a pinkish froth forms at the surface. The reaction appears earlier than Widal's (from the third day onwards), but is not so valuable, as it may occur in typhus, tuberculosis, malaria, and some other febrile diseases. Its absence is a strong argument against the existence of enteric.

Special Points of Treatment.—Here we have to do with a diseased alimentary canal: our treatment must be mainly dietetic.

Give no solid food, or that which would not readily pass through a fine sieve (WHITLA). Milk, *if it agrees*, arrowroot, beef-tea, and chicken-broth are the principal foods. The stools

should be daily inspected. If undigested curd is found, milk is being given too often, or the gastric function is impaired. It may then be given with lime-water or barley-water. Beef-tea should be sparingly used, lest it excite diarrhœa.

Alcohol is required mainly in the later stages, when the typhoid state has set in, and the heart is weak. Many cases do well without it altogether.

Medicinal.—*Do not be too officious.*

General principles hold good. If the diarrhœa becomes excessive give bismuth and opium, or lead acetate and morphia, or an enema of starch and opium. When the motions are very offensive intestinal antiseptics (calomel in small doses, salol, etc.) may be given.

If constipation be troublesome—enema of warm water, castor-oil. No irritating purge.

Hæmorrhage—opium, hypodermic injection of morphia, or ergotine, ice-bag to cæcum.

Perforation.—The main chance of recovery lies in early laparotomy and suture of the affected bowel. Every hour lost after the diagnosis is made, and the initial shock has passed off, increases the danger to life. Cases operated on within twenty-four hours may recover; later, recovery is very rare. In anticipation of operation, the gut may be paralysed with opium, or better, morphia.

Bed-sores—water-bed, cleanliness, stimulant and antiseptic lotions, dry dressings if the slough is large.

High fever—quinine and cold baths. The antipyrin group is dangerous to the heart.

The stools and urine must be carefully disinfected.

During convalescence the diet must be increased with the utmost caution, and the possibility of relapse must always be remembered.

TYPHUS FEVER.

An acute specific contagious fever, characterised by sudden onset, marked nervous symptoms, a maculated rash, and ending by crisis.

Ætiology.—Known as Jail fever, Ship fever, etc. ; is much more rare than formerly—occurs amongst the poor, in overcrowded and dirty districts, affects principally adults between twenty-five and thirty-six years of age. Very infectious, especially to doctors *and nurses*. No specific organism has yet been found. The virus appears to be present in the exhalations and excretions. Patients remain infective throughout the illness, and during convalescence.

Morbid Changes.—Those of intense fever, and blood-poisoning, plus the petechial rash. (See page 4). Leucocytosis is not constant.

Symptoms.—After a period of incubation the fever rapidly develops. The early symptoms are intense headache, nausea, and vomiting, with elevation of temperature. There may be rigors, pains all over the body, constipation, contracted pupils, thick-furred tongue, and rapid exhaustion. The skin gives off a mousy odour. Delirium is at first noisy. The face is congested, and the appearance dull, heavy, and apathetic. The rash appears on about the fifth day—first, on the abdomen, and the extensor aspects of the hands and wrists. Later it spreads over the trunk and extends to the extremities. As the fever progresses, the typhoid states comes on rapidly: the delirium becomes of the low muttering type—pupils, before contracted, may now be dilated; the grave complications tabulated may appear: retention of urine and paralysis of the sphincters; gangrene of the extremities: all these may appear and help to terminate the disease. If the patient does not succumb, the temperature falls, usually about the thirteenth day; profuse sweating, a critical diarrhœa, and an abundance of urates usually usher in a crisis; after which the patient gains strength rapidly, and may be, in a short time, in better health than before the attack. Second attacks are very rare.

The Rash—comes out rapidly about the fifth day. It shows—

1. A sub-cuticular mottling, *dusky-red* in colour (*measly eruption*).

2. Distinct papular rose spots, which tend to *become petechial*, and, therefore, disappear neither on pressure nor after death.

Complications.—

1. Early assumption of typhoid state.
2. Retention of urine.
3. Hyperpyrexia.
4. Gangrene of extremities, or bed-sores.
5. Bronchitis and broncho-pneumonia are the most common complications. They are often latent.
6. Parotid bubo, and pyæmic abscesses.
7. Thrombosis of femoral vein, more common than in typhoid.

It should be noted that, notwithstanding the severity of the cerebral symptoms, meningitis and nephritis are rare ; but albuminuria may be present.

Treatment.—*Special points to bear in mind.*—Whatever be the nature of the poison, oxygen has a remarkably destructive power over it ; therefore, a plentiful supply of air is indicated in this, perhaps more than in any other fever.

The nurse should be one who has either had the fever, or who has nursed typhus before. She should be specially instructed to note the tendency to *retention of urine and bed-sores*.

This fever seldom calls for lowering treatment, and frequently stimulants are required from the beginning. They should not be given as a matter of routine, but to meet special indications. Employ the general principles detailed in opening chapter. A good mixture is the following—

R	Acid Hydrobromic Dil.	℥ss.
	Quininæ Sulph.	℥ss.
	Aquam ad	℥viij.
						℥ss. 4 hor.

The contracted pupils and delirium suggest absence of excessive light, at least in the earlier stages. All noise must be avoided in the sickroom. Ice or cold affusion to the head is often useful. The food must be nourishing, and as plentiful

as the assimilative powers will admit. Milk, or milk and soda-water, and broths may be freely given, and when necessary, white of egg.

RELAPSING FEVER.

A specific infectious disease, occurring in epidemics, and characterised by terminating suddenly on the sixth or seventh day, but *followed by a relapse after an interval of a week*. The fever is always associated with specific organisms in the blood.

Ætiology.—Epidemics have a close connection with overcrowding and destitution. The disease is endemic in parts of India, epidemic in temperate climates, and especially in Ireland during periods of famine. Like typhus, it occurs amongst the poor and filthy, but is more associated with *poverty and famine* than filth. It attacks all ages, but males more than females. It is transmitted from person to person, and may be carried by fomites.

The Organism.—The *spirillum* or *spirochaeta Obermeieri* consists of a coiled thread, varying from $\frac{1}{500}$ to $\frac{1}{1500}$ of an inch in length. It is in constant movement of a lashing character. The spirilla tend to adhere around the red corpuscles. They are absent during the non-febrile interval, but return again when the relapse occurs. Just as they disappear before the crisis, they assume a granular appearance, probably due to degenerative changes. When they disappear from the blood, they accumulate in the spleen, and Metchnikoff asserts that they are then destroyed by phagocytosis.

The incubation period varies between one and twenty-one days.

Symptoms.—The fever is ushered in suddenly, with rigors, frontal headache, backache, and rapid elevation of temperature, which may reach, even on the first day, 104° . The pulse is very rapid, and respirations are also quickened. The tongue has a marked white fur, and there is much thirst. About the

fifth night the symptoms become greatly aggravated and the temperature may reach 107° or 108° ; the delirium is increased, and a fatal termination seems imminent, when a profuse sweating takes place, the bad symptoms rapidly abate, and the crisis is established. In a few hours the patient feels comparatively well; he is ravenously hungry, and has apparently fully recovered. Then in about a week, he is seized with symptoms similar to those of the first attack—usually the second attack, however, *runs a less severe course, and is of shorter duration than the first attack*, but sometimes the exhaustion is so profound, that the “typhoid” condition rapidly supervenes, and a fatal issue results. Two, or even more, relapses have occurred. Convalescence is usually slow. The disease is much less fatal than typhus.

Complications.—

Bronchitis, pneumonia, and pleurisy.

Dysenteric diarrhoea.

Severe jaundice.

Ophthalmia.

Hæmorrhages.

Special Points to note—

1. The spirochæta.
2. Absence of rash. (There may be petechial spots.)
3. The speedy, *apparent* recovery after the first attack.
4. The slow, *real* convalescence after a relapse.
5. Association of the fever with famine.

Morbid Anatomy.—Beyond the presence of the organisms, nothing special is seen in the tissues. The spleen and liver are, however, both enlarged, especially the former.

Treatment.—Relieve symptoms and maintain the general strength. Quinine and the mineral acids may be given. As many of the patients have been in a state of starvation, only fluid and easily digestible foods are at first allowable, and the supply must be very carefully increased.

VARIOLA or SMALL-POX.

A contagious, infectious disease, characterised by a rash, which usually appears on the third day, and runs through four stages, viz.:—(1) Papular; (2) Vesicular; (3) Pustular; (4) Drying up or Scabbing.

Ætiology.—Common to all ages and both sexes. The fœtus may be affected. Planted in a virgin soil it is very virulent. Its virulence is much modified by successful vaccination.

The contagion spreads by air, infected clothes, contact with people, and contact with the contents of the pustule, with scabs, or with the scales of desquamating skin. As the primary fever declines on the third day, when the rash appears, the patients frequently walk about and apply at hospital for advice respecting a “rash,” and they may thus infect all in the waiting-room. Of great importance to the State is the fact, that the virus may be given off from the dead.

No specific micro-organism has as yet been found. The virus is present in all secreta and excreta, and is given off from the lungs and skin. It is probable that the disease is contagious before the eruption appears.

Symptoms.—After twelve days’ incubation, the malaise of onset comes on. Most notable features—*frontal headache, pains in back, vomiting*. Temperature runs up rapidly, with all the phenomena of “fever.” On the third day fever usually declines, and the rash appears as a papule. Soon, however, the symptoms become worse and more pronounced than before, the fever increases, and on the ninth day, when the rash becomes pustular, the so-called secondary fever occurs, due to absorption of pus. The fever then becomes septic in type. Temperature oscillates. There may be severe rigors and a rapid assumption of the typhoid state. There is great swelling of the face—the eyes may be even closed up—and too frequently the patient dies; or the fever may subside, and the scabs commence to dry, falling off finally on the eighteenth or twentieth day, leaving a more or less pitted appearance.

The Specific Rash first appears on third day of fever, on the face, forehead, and scalp, as slightly raised red papules; feels *shotty* beneath the skin. It afterwards appears on the back of the wrists, then on the trunk and arms, and lastly on the legs. The mucous membranes are also affected.

Three days later (sixth day of fever) the "papules" become vesicular, at first clear and transparent, then turbid, depressed in the middle or *umbilicated*. Each vesicle is also *loculated*, that is, divided into compartments by delicate connective-tissue partitions derived from proliferation of the cells of the rete Malpighii.

Usually about five days later (eighth day of eruption), the vesicle becomes *purulent*, accompanied with an inflammatory ring round each vesicle, which causes great swelling and disfigurement of parts affected, and is attended with exacerbation of symptoms due to absorption—*i.e.*, secondary fever; then the pustule begins to dry; a black-brown scab forms and drops off, leaving a depressed scar as before mentioned.

Varieties.—

1. Modified, as seen occasionally after vaccination.
The initial fever is usually slight, and the rash scanty. The papules vesicate on the fifth day, and dry up in a day or two. There is no secondary fever.
2. *Simple* or *discrete*—the pocks being few.
3. *Confluent*—where pocks run into one another.
Initial fever is severe, the eruption appears early, and the temperature does not fall on the third day. Death takes place on the ninth day in unvaccinated subjects, on the eleventh in the vaccinated, as a rule.
4. *Hæmorrhagic* or *malignant*. The specific rash is often preceded by one or other of the malignant rashes mentioned below.
5. *Corymbosc*, where pocks cluster together like grapes.

Other varieties have been tabulated, but they only confuse the student and serve no practical purpose.

How does the hæmorrhagic form influence the clinical course?

1. The patient may be so overwhelmed with the poison that he may die as early as the second day.
2. The hæmorrhage may appear later, and prevent the ordinary stages or course of the rash.
3. In any case the prognosis becomes more grave, and the patient may be carried off by hæmorrhages from mucous membrane, nose, lung, kidneys, etc.

Next to the hæmorrhagic, of course, the confluent is the most severe form—*fever does not fall on the third day*. The fever becomes more severe, the skin enormously swollen, and all the symptoms tend to a low type.

Complications.—

1. Pyæmic abscesses and albuminuria. ✓
2. Suppurative keratitis, with ulceration of cornea.
3. Ulceration of pharynx, or larynx.
4. Bronchitis, septic pneumonia, and pleurisy.
5. Hæmorrhage of *all* kinds.
6. Gangrene, usually of penis.
7. Purulent arthritis.

Sequelæ.—

Otitis media.
Abscess of bones.
Deep pitting.
Blindness.
Peripheral neuritis.

Pathology.—Tissues generally—like those of fever. (See page 4.)

Rash.—1. Papular stage.

- (1) Proliferation of cells of rete mucosum.
- (2) Œdema, cell coagulation, and central necrosis.
- (3) Compression of other cells to form trabeculæ.

2. Formation of spaces containing—

Serum.	}	Vesicular stage.
Fibrin.		
Leucocytes.		

The umbilication corresponds to the area of primary necrosis. Its connection with the site of a hair follicle is accidental.

3. Degeneration into pus of vesicular contents—papillæ of true skin are now very swollen and infiltrated with leucocytes—pustular stage. The pustules contain streptococci and staphylococcus pyogenes.

There is marked leucocytosis, the lymphocytes being chiefly increased, the polynuclear leucocytes diminished.

Initial Rashes.—The specific rash may be preceded by other rashes (which may appear on second day)—

1. Hæmorrhagic or petechial—occupies lower half of abdomen, and extends downwards in a triangular form to an apex in Scarpa's triangle.
2. Petechio-erythematous—of wider distribution, extending up sides of chest.
3. Erythematous form—similar to scarlet fever or measles. If this happen, the true rash when it occurs seems to spare the site of the initial rash.

The erythematous form indicates a slight attack, and the prognosis is gravest in the petechio-erythematous.

Treatment. — Vaccination or re-vaccination carried out within three days of exposure to infection will either prevent or modify the ensuing attack. Later it is useless. All those in contact with the patient must be vaccinated, and rigid isolation must be insisted upon. There is no specific drug, and treatment must be carried out on general principles.

As small-pox is infective even in the prodromal stage, patients with suspicious symptoms should be at once isolated.

Special points are—

1. Cut the hair and beard close.
2. Sponge daily with tepid water, or in confluent cases use the warm bath.
3. Ointments for the prevention of itching or pitting are useless. Warm lead lotion may be of service. The windows may be covered with red curtains to exclude the ultra-violet rays.
4. Cover the eyelids with cold compresses, to reduce œdema. Where the cornea is ulcerated, a single application of the yellow oxide of mercury to the ulcer should be followed by the frequent use of a lotion of Ext. Belladonnæ gr. x - ʒj, applied warm.
5. Treat throat complications by ice and antiseptic sprays.
6. Quinine in 5-gr. doses and Tr. Fer. Perchlor., ℥ xx - xxx are the best internal antiseptics.
7. In hæmorrhagic cases, stimulate freely.

VACCINATION IN MAN.

Jenner first inoculated cow-pox, with lymph obtained from pustules on the udders of cows. The contents of the vesicle so set up may be used in arm-to-arm vaccination. To avoid the risk of the transmission of syphilis, calf lymph is now generally employed.

After inoculation—

A papule forms on the second or third day.

It becomes an umbilicated vesicle on the fifth or sixth day.

It becomes purulent and an areola forms round it on the ninth day.

It dries up, and the scab falls at the end of the third week, leaving a permanent scar.

The neighbouring lymphatics become enlarged. After inoculation the arm usually swells and more or less fever is occasioned.

British law requires all healthy children to be vaccinated before the age of six months.

In ordinary circumstances, vaccination should be postponed if the child is feverish, if it has any specific infectious disease, any cutaneous disease, especially eczema, or if it has diarrhœa. Where there has been exposure to small-pox it must be done at all risks, except in the presence of serious acute disease.

VARICELLA or CHICKEN-POX.

An eruptive fever, occurring principally amongst children, and characterised by a rash at first papular, but rapidly becoming vesicular, which appears on the first day, and is repeated in successive crops during the next two or three days.

Ætiology.—Epidemics are very common, and the disease once it starts usually spreads with great rapidity. Sporadic cases, however, are not uncommon. No specific organism has been isolated. The disease is contagious, but not inoculable. One attack confers immunity. The usual incubation period is fourteen days.

The Rash consists of small papular rose-spots, of very varying number, which within twenty-four hours become raised vesicles, containing either transparent or turbid fluid. It usually appears first on the neck and chest, but quickly spreads over the entire body. Each crop requires from five to six days to complete itself, and thus the illness lasts rather over a week. Modified spots may appear on the palate, buccal mucous membrane, and tongue.

It differs from small-pox in—

1. Not being umbilicated or loculated.
2. Having no inflammatory areola around the vesicles.
3. Appearing on the first instead of the third day.
4. Being vesicular almost from the beginning.
5. The vesicles usually beginning to dry up as brownish scabs on the fourth day, leaving no scarring or pitting.

6. In small-pox the fever declines when the eruption appears ; not so in chicken-pox.

Symptoms are not severe. There is usually slight fever and fretfulness, with a very furred tongue and rarely vomiting.

Complications—

1. *Severe itching*, causing the child to scratch, producing deep scars, or even ulceration.
2. Gangrene in debilitated children around vesicles (*varicella gangrænosa*). (I have often seen this in children with congenital syphilis [A. W.] It also occurs in tubercular children.)
3. Infantile paralysis (very rare).

Treatment.—A gentle saline purge, careful dieting, and where there is much itching, soothing lotions on lint—morphine and lead.

SCARLATINA or SCARLET FEVER.

An acute infectious disease, characterised by fever, sore throat, and an erythematous rash, followed during convalescence by free desquamation, and often complicated by otitis, nephritis, or arthritis.

Ætiology.—Epidemics prevail at all seasons, but are most prominent during the latter half of the year. The disease is commonest in children between two and ten years old, though adults are often attacked. It is usually spread by direct communication, but may be carried by infected clothing or by a third person, the virus remaining active for a long time. It may also be spread by milk, which has been contaminated either by man or by the cow. Cows and swine both suffer from a disease which may produce scarlet fever in man.

Klein has described a specific organism, the streptococcus scarlatinæ, and Class has isolated a diplococcus, for which he also claims specificity. The question is as yet undecided.

The usual *incubation period* is from two to three days.

One attack generally confers immunity, but second and even third attacks may occur.

Symptoms.—The principal features of the onset are—vomiting, rheumatic pains, soreness of throat, fever, and headache. In infants, convulsions are not uncommon. Soon the throat becomes very sore, the neck feels stiff, and the glands at the angle of the jaw are swollen.

On the first or second day the rash comes out, first on the chest, then rapidly spreads over the face, body, and extremities. Its appearance may be delayed for a day or two.

As the fever progresses, the throat symptoms become more severe; the tonsils approach the middle line, and yellowish points appear. These may coalesce and form a “patch” resembling that of diphtheria. Other severe complications (see tabulated list) often arise and bring about a fatal issue; or the temperature may fall gradually with the fading of the rash, and a slow convalescence is gradually established.

The Tongue is at first covered with thick white fur, with papillæ projecting—*i.e.*, the white strawberry tongue; later, the fur peels off, leaving the typical red strawberry appearance.

The Urine.—There may be ordinary febrile albuminuria, even where there is no nephritis. The diazo-reaction may be present.

The Blood.—There is generally a marked leucocytosis, the polynuclear cells being chiefly affected.

The Rash appears on the second day, first as a scarlet blush, but if looked into carefully it is seen to consist of small red spots, surrounded by a diffuse erythema.

The severity of the rash varies; it may be absent or only present in the “flexures” of limbs as bright red lines. Diagnosis is then often difficult, but later on desquamation settles the question.

Desquamation begins as early as the sixth day; it may be only to the extent of roughness, or the epidermis may peel off

in large flakes ; time occupied = 4 to 8 weeks. It continues longest on the palms and soles.

Complications.—

1. Scarlatinal arthritis, in the forms of—(a) scarlatinal rheumatism, about the end of the first week ; (b) suppuration of one or two large joints ; (c) general pyæmic infection.

2. Endocarditis and broncho-pneumonia.

3. Ear troubles are exceedingly common. The suppurative process going on in the middle ear may cause—

- (1) Perforation of the drum.
- (2) Permanent deafness, through suppuration extending to internal ear (not common).
- (3) Facial paralysis.
- (4) Meningitis.

4. Extensive suppuration of glands, particularly the cervical glands.

5. *Scarlatinal nephritis* is, however, more of a sequela, as it most often begins about the second or third week of illness, when the skin is freely desquamating. The symptoms are—

- (1) High-coloured or smoky albuminous urine.
- (2) Presence of epithelial casts, blood casts, and blood corpuscles.
- (3) Dropsy of eyelids and ankles, but the dropsy may extend all over the body.
- (4) In grave cases uræmia may occur, and lead to convulsions, coma, and death.

Varieties.—

1. *Scarlatina Benigna*, or Simple Scarlet Fever. The rash fades in a few days, and the temperature is normal within a week.

Scarlatina Anginosa, or Septic Scarlet Fever. The throat symptoms are severe and a membranous exudation forms upon the tonsils and palate, leading to necrosis of the tissues of the

throat, with intense fœtor. There is extensive sloughing, which may cause perforation of the ascending pharyngeal artery or even the common carotid. The rash lasts a week or more, and the temperature may not be quite normal for several weeks.

3. *Scarlatina Maligna*, or Toxic Scarlet Fever. The symptoms are grave from the beginning, and the rash abundant, but sore throat is not marked. Delirium passes into coma, and death takes place within a week.

4. *The hæmorrhagic form*, in which extensive hæmorrhages occur (epistaxis, hæmaturia, etc.), is now very rare.

Special Points in Scarlet Fever.—The skin is peculiarly hot and pungent. Note the frequent tendency to attack serous membranes.

The nephritis is usually late, the explanation being not only that the kidneys have been overworked, in eliminating the products of the fever, but probably that the scarlet fever poison has a peculiarly irritating effect on the renal tubules. It may follow either mild or severe cases, and there is no reason to suppose that it is due to "catching cold." It very seldom leads to chronic affection of the kidney. It tends to affect the glomeruli more than ordinary nephritis, and hence is often called glomerulo-nephritis.

Treatment.—The same general principles as those detailed in opening chapter apply here.

Special Points are—

1. *Serum treatment.*—Serum from convalescents, injected in doses up to 20 cc., has sometimes proved successful. Various antistreptococcic sera have also been employed.

2. The danger of spreading the disease is greatest during the desquamative period. Isolation must therefore be kept up for at least six weeks from the onset, or till all desquamation and discharges (nasal, aural, etc.) have ceased.

3. A minimum amount of *nitrogenous* food, to avoid irritation of the kidneys.

4. Daily toilet, tepid sponging, or tepid baths.

5. Inunction of oily antiseptic preparations into the skin, to prevent dissemination of the desquamating scales.

6. Examine the urine daily for signs of nephritis.

Complications.—*Arthritis* demands warm and sedative applications. Wrap joints in cotton wool; alkalies are better than salicylates, as the latter tend to irritate the kidneys.

Throat.—Ice to suck; antiseptic sprays (chlorine water, etc.); glycerine of carbolic acid; warm applications externally.

For the severe types.—Avoid caustic applications; relieve pain by cocaine solutions; internally perchloride of iron, with free administration of ammonia; quinine, either alone or with perchloride of iron. Stimulants may also be required. Nasal feeding often gives much relief.

Nephritis.—Milk diet, hydragogue purgatives (avoid mercurials), hot-air baths, hot packs, etc., according to the severity of the dropsy.

MORBILLI or MEASLES.

An eruptive fever ushered in with coryza.

Ætiology.—Extremely infectious. Contagion is communicated by the nasal secretions and breath; also by fomites, or by a third person. Epidemics occur oftenest in spring and autumn. The disease is infectious during incubation, but most so when the rash is out. The incubation period is from eight to twelve days. No specific organism is known. One attack does not confer complete immunity.

Symptoms.—*First*, are those of catarrh—eyes water, conjunctivæ become suffused, discharge from nose, and bronchitic symptoms. Often temperature falls on second day, though not to the normal level, but *rapidly rises with the eruption* of the rash on the fourth day. As the fever progresses the bronchial troubles increase, and the various complications arise.

Koplik's spots very often appear on the buccal mucous membrane, within the lips, and even on the gums. They are small rounded red spots with a bluish central area. They precede the true rash by one to three days, and as they are very constant, they are of importance to an early diagnosis.

The Rash.—First appears on *fourth* day at the roots of the hair, and on forehead and face. The rash consists of raised, dark-red papules, coalescing into crescentic patches, and giving a velvety feel to the touch. There is usually slight subcutaneous œdema, and the child presents a peculiar, blotchy, swollen appearance. After extending to the neck, trunk, and limbs, the rash fades in about three days, leaving a slightly brown-stained appearance which soon passes away. There may be slight, branny desquamation, *but it is always slight*, and best seen on the face. The rash differs from that of scarlet fever by—

1. Appearing on the fourth instead of the second day.
2. Being darker in colour.
3. The velvety feel.
4. The crescentic arrangement.
5. Manner of spreading, but diagnosis is sometimes difficult.
6. The subcutaneous œdema.

The rash may be slightly hæmorrhagic, even in cases which are not severe.

In simple cases convalescence is usually complete in eighteen days.

Complications.—

1. The extension of bronchial catarrh, causing capillary bronchitis and collapse of the lung, or pneumonia either catarrhal or croupous in nature. Dr. John Playfair notes a frequency of *croupous* pneumonia and bronchitis in the same lung. In children, laryngitis is often formidable.
2. Purulent ophthalmia, etc.
3. Otitis media; swollen cervical glands.

4. Gangrene of skin or vulva.
5. Co-existent diphtheria.
6. Nausea or vomiting ; diarrhœa.

The sequelæ are very numerous, and whooping-cough is frequent among them. Tuberculosis is also liable to follow, and occasionally chronic endocarditis.

Varieties.—

1. Simple.
2. Malignant, comprising
 - (a) Hæmorrhagic measles, characterised like all other hæmorrhagic forms of fever by bleeding from mucous surfaces, hæmaturia, and an early assumption of the typhoid state. This is a rare form.
 - (b) Adynamic measles, in which the symptoms are grave from the outset, but without hæmorrhages, and the typhoid state is early present.
 - (c) Measles, complicated with diphtheria of the fauces.

Pathology.—The first of the pathological changes is a specific catarrhal inflammation of the mucous membranes of the respiratory and intestinal tracts. The papule, according to Unna, is due to œdema of the cutis and hypoderm, not, as in small-pox, to epithelial changes. Cellular exudation is almost completely absent. There is no leucocytosis, unless, it may be, before the eruptive stage.

Treatment.—General principles hold good. Owing to the condition of the respiratory organs, the greatest care must be taken to avoid chill until all catarrhal symptoms have passed away. Simple cases need little more than careful nursing. In convalescence cod-liver oil, Syr. Ferri Iodid., Syr. Hypophosph. Co., and similar drugs are indicated. Cases should be at once isolated, and may be released at the end of three weeks after thorough disinfection, if all catarrhal symptoms have gone.

RÖTHELN—GERMAN MEASLES— RUBELLA.

An infectious eruptive fever, not attended by catarrh, but accompanied by swelling of the cervical glands.

Ætiology.—Rubella was formerly thought to be a mixture of scarlet fever and measles, but is now held to be a separate disease. It spreads by contagion, and epidemics are most common in the spring. The average incubation period is fourteen days. One attack does not confer immunity.

Symptoms.—Those of slight headache, sore throat, and swollen cervical and occipital glands; *rarely are there severe complications*. Many patients do not feel ill at all, but Cheadle reports some severe cases attended with albuminuria.

Rash.—Round or oval, slightly raised, pinkish-red spots, discrete at first, but afterwards coalescing. It appears on the first or second day, begins on the face, and extends to the body and limbs while fading on the face. Fever and rash subside together on the third day.

Diagnosis.—From Measles by—

1. Short prodromal stage.
2. Absence of the dark colour and crescentic form of the measles rash.
3. Absence of Koplik's spots.

From Scarlet Fever by—

1. Large size of spots.
2. Absence of severe symptoms and desquamation.

Treatment.—General principles. A child exposed to infection should not be allowed to mix with others for twenty days. A convalescent is free from infection in not less than ten days from the appearance of the rash.

PERTUSSIS or WHOOPING-COUGH.

A specific infectious disease, affecting the respiratory organs, and attended with a peculiar paroxysmal cough and whoop.

Ætiology.—One attack procures future immunity. It is highly contagious from person to person, and is also spread by fomites; one single case will frequently cause the infection of a *whole* village or town. The virus is chiefly disseminated in the sputum. There can be no doubt that the disease is due to an organism, the true nature of which has not yet been determined, although both *amœbæ* and bacteria have been described by various observers.

The disease is most infectious in the first week, and becomes gradually less so. Patients may be considered non-infectious five weeks after the first whoop. The average incubation period is about eight days. Epidemics occur chiefly in spring and early summer and are often closely associated with measles.

Pathology.—The spasmodic nature of the cough is due to implication of the nervous system, probably the result of toxic invasion. Leucocytosis is well marked, the lymphocytes being chiefly involved. *Post-mortem* evidences of bronchial catarrh, and of pulmonary or other complications, may be found.

Symptoms.—The disease is usually divided into three stages—

1. Stage of invasion (catarrhal stage).
2. Paroxysmal or spasmodic stage.
3. Period of decline.

1. *Stage of Invasion.*—The onset is either insidious or well marked, the temperature rising smartly to 100°-102°. The symptoms are merely those of bronchial catarrh with coryza, and last from seven to ten days.

2. *Spasmodic or Whooping Stage.*—The paroxysm consists of a series of short coughs or expiratory puffs, with no intervening inspiration till about fifteen or more expulsive efforts have been made in about seven seconds. Then occurs a deep, prolonged inspiration, attended with the characteristic *whoop*; a second bout of short coughs succeeds with another whoop, and after three or four such sequences, a little plug of mucus is expelled; or more frequently vomiting takes place, and the child is apparently well until the next paroxysm.

During the severe cough the patient is perfectly helpless, and when the paroxysms are very violent we may get—

1. Hæmorrhages from the nose, frænum of the tongue, under the conjunctivæ, or even in the brain.
2. Ulceration of the frænum
3. Collapse of the lung.
4. Convulsions in infants.
5. Fatal asphyxia (rare).

The appearance of the child soon becomes puffy or bloated. The whoop is due to partial closure of the glottis. The closure is considered by some to be “reflex spasm” in nature. Others say it is a mere passive approximation of the glottis. There may be as many as forty attacks in the twenty-four hours, but they range from four upwards; this stage lasts three to six weeks. Sometimes the “whoop” is delayed in its appearance; difficulty will be then experienced in diagnosis, and no general law can be laid down. Niemeyer says, “If a child has a violent, *prolonged cough* attended with *vomiting*, suspect and treat as if it were whooping-cough.” I am certain this is sound advice, borne out by experience of two epidemics I saw in Monmouthshire (A.W.). During the expiratory spasm, the percussion note is dull. Râles may be heard over the chest throughout the paroxysm. In the intervals there are usually no physical signs.

3. *Period of Decline*.—This stage is marked by a gradual decrease in the number of paroxysms, the disease lasting from six weeks to two months or more. Convalescence is slow, and may be protracted over several months. At this stage patients are particularly liable to tuberculosis.

Complications.—Besides those mentioned as possibly occurring during the paroxysm there may be—

1. Bronchitis, capillary bronchitis or broncho-pneumonia, rarely subcutaneous emphysema from rupture of air-cells.
2. Convulsions, and more rarely cerebral paralysis.
3. Cardiac strain ending in valvular disease.

Treatment.—Prompt isolation ; and to begin with, a simple saline with paregoric. When the whoop develops we must try anti-spasmodics ; bromoform, belladonna, chloral, or antipyrin ; others advise in addition—quinine, emetics, swabbing the throat with a 2 per cent solution of resorcin, etc. The number of drugs recommended by various authorities shows that none of them is specific. Even the anti-spasmodics have little influence on the paroxysms unless given in such doses as to be otherwise harmful. Inhalations of creosote, carbolic acid, sulphurous acid, etc., have proved useful in many cases.

Adequate ventilation and avoidance of cold and draughts are very important. In warm weather the child should be much in the open air, unless serious complications are present.

If recovery be tedious, *change of air*, cod-liver oil, and Easton's syrup.

Special points to be noticed—

1. In *infants*, tendency to convulsions.

2. In *older children*, capillary bronchitis, causing—

(1) Collapse of lung.

(2) Deformity of chest (pigeon shape).

(3) Children exposed to infection should be disinfected and isolated for at least three weeks, as the disease cannot be diagnosed in the catarrhal stage.

MUMPS or EPIDEMIC PAROTITIS.

An acute infectious disease, characterised by inflammation of the salivary glands, especially the parotid gland, with a tendency to metastatic inflammation of testes in males, or breasts in females.

Ætiology.—Is frequently contagious, especially in schools, and may be curiously localised in one district. The exciting cause is possibly a streptococcus discovered by Michaelis, and closely resembling the gonococcus : but the disease has not as yet been reproduced in animals. Children from four to twelve years of age, and young adults, are most often attacked. The disease is infectious even before the glands are affected, and

for two to three weeks afterwards. The incubation period lasts from two to three weeks. Second attacks are rare.

Anatomy.—The inflammation is interstitial rather than parenchymatous, the connective tissue in and around the glands being attacked, while the acini escape. It rarely goes on to suppuration.

Symptoms.—After a period of incubation, pain is felt under one ear, with stiffness or soreness of neck and jaw. This is usually accompanied by smart fever (101° to 103° or 104°) which subsides on the third or fourth day. The swelling first appears in the hollow between the angle of the jaw and the mastoid process, gradually extends, and may involve the sub-maxillary and sub-lingual glands. It is elastic and tender on pressure, but not fluctuant. In about two days the *other side* may undergo the same changes—both swellings form a “collar,” giving the child a ludicrous appearance. Oftener the swelling is confined to one side, or appears on the other when the first is going down. Deglutition and mastication are often very painful, the breath is foul, and tongue very furred. In about nine days the swelling resolves, and rapid improvement takes place. The glands seldom suppurate.

Complications.—

1. Unilateral orchitis occurs as the glandular affection subsides, or later, in nearly one-third of the cases seen in adult males. It subsides quickly, but may lead to atrophy of the testicle. Epididymitis is uncommon.
2. In the female, mastitis and œdema of the vulva may occur.
3. Meningitis is a rare complication.
4. Endocarditis has also been recorded.

Sequelæ.—After severe cases the following have happened—

1. Permanent deafness, following otitis media or interna.
2. Arthritis, rarely purulent.

Treatment.—A simple saline purge, hot fomentations, and an antiseptic wash for the mouth are all that is usually required. Treat complications as they arise.

INFLUENZA.

An acute specific infectious disease characterised by fever, by symptoms affecting mainly the respiratory, digestive, and nervous systems, and by prolonged prostration following upon defervescence.

Ætiology.—Epidemics are most frequent in the winter months, when respiratory diseases are prevalent. Adults between the ages of twenty and forty are oftenest attacked, young children and the aged less often. Sedentary occupations predispose to the disease.

The *bacillus influenzae*, discovered by Pfeiffer in 1892, is a very small rod-shaped organism ($0.2\ \mu$ wide, $0.5\ \mu$ long), occurring singly or in pairs or short chains. It is non-mobile, and does not bear spores. It can be cultivated on blood-agar, on which it forms very minute colonies.

It is found in the respiratory secretions, and less commonly in the lung, heart, central nervous system, etc., but very rarely in the blood. Contagion is conveyed by the moist secretions of the nasal and bronchial mucosa.

The incubation-period is from two to six days. One attack confers no immunity.

Pathology.—The characteristic changes are—

1. Inflammatory swelling of the nose and neighbouring sinuses.
2. Hyperæmia of the trachea and bronchi, the surface of which is covered with tenacious muco-pus.
3. In fatal cases patches of broncho-pneumonia.
4. When the central nervous system is involved, hyperæmia of the meninges of brain and cord.

Symptoms.—The onset is sudden. There are severe frontal headache and backache, pains in the bones, and marked weakness. The temperature rises smartly to 102° or even 104° . Coryza and catarrh of the upper air-passages are present, and the larynx or pharynx is often involved. In mild cases convalescence sets in in a few days, but a feeling of prostration

persists for a considerable time. In severer cases, after the first few days, the disease assumes one of three types :

1. *Respiratory type*, in which bronchitis, broncho-pneumonia, or croupous pneumonia develops.
2. *Gastro-intestinal type*.—Epigastric pain, vomiting, diarrhoea, and anorexia, associated sometimes with jaundice and enlargement of the spleen, are the commonest symptoms.
3. *Nervous type*.—The initial pains are more severe, and after defervescence the heart becomes slow or irregular, and there is sometimes anginoid pain. Great depression and insomnia follow. In grave cases there may be coma or delirium. Meningitis or encephalitis may be found post mortem.

Complications and Sequelæ.—

1. Any form of nervous disorder may follow or complicate the nervous or the other types of influenza :— hæmorrhage, embolism, epilepsy, insanity : myelitis or degeneration of the cord : neuritis, local or general, and neuralgia.
2. Albuminuria, sometimes leading to chronic nephritis.
3. Pulmonary and cardiac complications.
4. Arthritic pains.
5. Accidental cutaneous rashes. Influenza has no proper rash.
6. Hyperpyrexia.

Prognosis in uncomplicated cases is good, except in the elderly, to whom the disease proves very fatal, usually from the supervention of pneumonia. Quiescent chronic diseases are apt to be kindled into activity by an intercurrent attack. Relapses are common.

Treatment.—Rest in bed should be insisted on even in the mild cases, and continued for a day or two after the temperature has become normal, in order to avoid the risk of relapses or complications. Great care must be taken during convalescence, especially in the elderly.

The pains of the first stage are best relieved by salicylate of soda, or where headache is severe by phenacetin. Quinine is also useful at this stage. Diarrhœa may be checked by warmth to the abdomen and by fluid foods; bismuth and sedatives are of little use. For insomnia bromides may be given, particularly the bromide of ammonia. In convalescence tonics, and especially strychnine, are of importance.

Complications must be treated as they arise.

EPIDEMIC CEREBRO-SPINAL MENINGITIS.

An acute specific fever characterised by inflammation of the cerebro-spinal meninges, and clinically by sudden invasion and extreme nervous shock, attended by painful contractions of muscles, cutaneous eruptions, and various grave nervous complications.

Ætiology.—The disease occurs both in limited epidemics and sporadically. It affects children and young adults chiefly. Infection occurs chiefly through the nasal passages. A recent report by the city bacteriologist of Glasgow states that the meningococcus was found in the naso-pharynx of 20 out of 81 “contacts,” and in the naso-pharynx of patients and convalescents, in one instance as long as the seventy-fifth day after the onset. The infection may thus “be carried by individuals who offer no evidence of its presence, and may remain a long time in the throats of individuals stricken with the disease, or who have recovered from it.” Cases are recorded of a similar disease in horses, during its epidemic prevalence in man. Extreme fatigue appears to increase the liability.

The micro-organism is the *Diplococcus intracellularis meningitidis* of Weichselbaum, which is found mainly within the polynuclear leucocytes of the exudate. It is aerobic, and can be grown on glycerin-agar or blood-serum. It stains easily with ordinary stains, and is decolourised by Gram’s method.

Symptoms.—The invasion is sudden; there are intolerable pain in the back of the head and neck, extending later down the spine, vertigo, vomiting, and noisy delirium, which is

succeeded by apathy or even by coma. There is often intense hyperæsthesia. The *head is strongly retracted*, and the limbs and trunk are rigid, causing opisthotonos or orthotonos. Tetanic convulsions may occur. The temperature is sometimes little raised, in other cases it may reach 104° or 106°. The pulse varies similarly. The skin may therefore be either cold or warm. Cutaneous eruptions may appear from the first to the third day, about which time consciousness returns, and there may be a transient improvement in the symptoms. They are soon aggravated, the fever rises, and diarrhœa sets in. There may be hemiplegic paralysis of one or more limbs. Exhaustion and emaciation are rapid, and typhoid state is early established, anæsthesia takes the place of hyperæsthesia, and the patient quickly succumbs. If he survive, the symptoms subside only slowly, and convalescence is long and perilous.

Cutaneous eruptions.—These are usually of one of two forms :—

1. *Herpes facialis et cervicalis*, and *herpes zoster*. The distribution is often wide, and the eruption need not be limited to one side.
2. *A purpuric or petechial rash* (hence the American name of “spotted fever”) usually beginning on the legs. It may involve the entire surface.

Erythematous and pemphigoid rashes occasionally occur.

Leucocytosis or increase in the number of leucocytes (25,000 to 40,000 per cmm.) is constantly present.

In the so-called *malignant* form of the disease, death takes place in from a few hours to three or four days from the onset.

Complications.—

1. Pleurisy, pericarditis, pneumonia.
2. Purulent effusions into joints.
3. Optic neuritis, purulent keratitis, or panophthalmia (usually in the right eye).
4. Deafness, sometimes permanent.

Special Points to note in this meningitis are—

The marked rigidity, and tendency to opisthotonos or orthotonos.

The varied skin affections.

The sudden onset and extremely rapid course.

Morbid Anatomy.—In rapidly fatal cases there may be nothing more than intense congestion of the meninges. In those of longer duration there is an abundant whitish or yellowish fibrino-purulent deposit at the base of the brain and in the lumbar portion of the cord. The membranes are much thickened; the ventricles are dilated and contain pus or turbid fluid. The veins of Galen may be thrombosed, causing hydrocephalus.

Diagnosis.—Apart from the symptoms, there are two important aids to diagnosis.—

1. *Kernig's sign.*—If the thigh be flexed at right angles to the abdomen, the leg cannot be fully extended upon the thigh, as it can in health, owing to strong contraction of the flexors.

2. *Lumbar puncture.*—The skin over the lumbar region being anæsthetised, the patient is laid on the left side with the back bent and the knees drawn up. The third lumbar spine is found, and a small aspirating needle thrust deeply upwards and inwards in the third interspace, till the subarachnoid space is reached. The cerebro-spinal fluid, in meningitis turbid or purulent, can thus be collected and examined for the presence of the diplococcus. With ordinary precautions, this operation is perfectly safe.

Treatment.—When there is marked collapse in the early stages, warmth, stimulants, and mustard may be used; later, the indications are to subdue the spinal irritation and to relieve pain. To meet the former, atropine or belladonna, bromides or ergot may be given; for the latter, opium is mainly to be relied upon, and must often be freely used. Leeches to the nape of the neck, and ice to the head are also useful, and blisters may be applied along the spine. Repeated withdrawal of cerebro-spinal fluid by lumbar puncture is often of use, at

least in relieving symptoms due to pressure, and may lead to cure. When the fluid is purulent, antiseptic solutions (10 cc. of a 1 per cent lysol solution) may be injected after its removal. Serum treatment is at present on its trial.

The nasal passages must be disinfected, and nasal discharges burnt.

The period of quarantine for contacts is one week.

DIPHTHERIA.

An acute specific infectious disease attended with grave throat symptoms and general symptoms, and the formation of a false membrane or fibrinous exudation on mucous and abraded surfaces, and often followed by paralysis in various situations.

Ætiology.—The disease is endemic in the larger towns, and becomes often epidemic at various seasons. It is highly contagious, and the poison is very concentrated in the *pharyngeal secretion*; it is very fatal to doctors and nurses. The spluttering of secretion into the face whilst examining or swabbing out the patient's throat accounts for this great tendency to attack the attendants. The bacillus may be conveyed to the healthy from the discharges of the sick, from the throats of convalescents in whom the bacillus persists, and from the throats or clothing of healthy people where it has found an accidental lodging. Schools are a fruitful source of infection. The virus is extremely tenacious of life.

Epidemics can often be traced to spreading through—

1. Contamination of milk supply.
2. Exposure to sewage effluvia, owing to the sore throat so produced preparing the way for an attack.
3. The prevalence of sore throat *prior to epidemic*—i.e., formation of suitable soil for the germ.

There is no evidence that it has ever been conveyed by polluted water.

The disease is most prevalent in the third and fourth quarters of the year. Children between the ages of two and

ten are oftenest attacked, but it is quite common in adults. Individual susceptibility varies greatly, the organism being often found in the throats of healthy people, especially when they have been associated with patients. Other diseases, such as scarlet fever and measles, weakening both the fauces and the system, render the liability greater. One attack does not confer immunity. The incubation period varies from two to seven days.

Pathology.—*Specific Germ*—It is known as the Klebs-Loeffler bacillus. It is non-mobile, slightly bent, and knobbed; it stains with ordinary dyes, and is not decolourised by Gram's method. It grows on all ordinary media and multiplies readily in milk. It is found in the false membrane associated with other germs, such as streptococci, and staphylococci ("mixed infection"). It does not penetrate the mucosa. Diphtheria is, therefore, primarily a local disease, the constitutional symptoms being produced by the absorption of toxins. The false membrane shows—

1. A heavy network of epithelium.
2. Fibrinous threads entangling masses of leucocytes, and proliferated connective-tissue corpuscles.
3. Colonies of micrococci with the Klebs-Loeffler bacilli.
4. Granular debris.

The local changes, as described by Oertel, are as follows:—The poison first induces a necrosis of the cells with which it comes in contact; the superficial epithelium thus first disappears. The deeper cells become similarly affected, and a zone of inflammation forms around the dead cells; the membrane thus is really a mass of dead cells undergoing hyaline degeneration, and mingled with fibrin, and it presents the peculiar laminated appearance considered characteristic. The neighbouring lymphatic glands become much enlarged.

Heart.—Fatty or hyaline degeneration of the myocardium is extremely common.

Kidneys.—Either the ordinary cloudy swelling or intense and acute nephritis may be present. The latter is not common.

Nervous System.—At a later stage, the peripheral nerves supplying the parts that may be paralysed are found in a state of partial degeneration of their medullated fibres (toxic neuritis).

The other visceral changes are similar to those of any other intense or malignant fever.

Very marked leucocytosis is common. It affects chiefly the polynuclear cells.

Symptoms.—After an incubation of two to seven days a general malaise sets in; there are *slight* fever, stiffness of the neck, and swelling of the glands at the angles of the jaws. The soft palate is deeply congested, and whitish patches soon appear, first on the fauces; these patches coalesce, become “wash-leather-like” in colour, and the false membrane is fully formed. It may extend *all over* the fauces, or may begin on the tonsils first, and creeping forward surround the uvula like a finger of a glove. The membrane is at first easily stripped, but soon re-forms; then it becomes firmly adherent, and if torn away leaves a bleeding surface. If left alone it may slough off.

The glands in the neighbourhood enlarge, but do not usually suppurate.

Anæmia and weakness set in early, and are rapidly progressive. The pulse is very frequent and feeble.

The temperature varies—it may be 103° or higher, but is usually from 100° to 102° , and indeed sometimes *subnormal*. This is peculiar, as most acute throat affections have high temperatures. The exudation may now extend in any direction, upwards or downwards, into—

1. The naso-pharynx, attended with coryza, regurgitation through the nose, epistaxis, and a nasal twang of voice.

2. The larynx; symptoms are stridor, brassy cough, great dyspnoea, and sucking in of the intercostal spaces. The membrane may *first appear in the larynx* (“diphtheritic croup”), and hence the symptoms just mentioned may be present before any characteristic appearances in the fauces.

It may remain limited to the larynx, or may extend upwards to the pharynx or downwards to the bronchi.

3. The bronchi, with all the symptoms of severe capillary bronchitis. The membrane after extending to the first bifurcation of the bronchus speedily becomes purulent.

4. The middle ear, along the Eustachian tube.

In many instances the false membrane remains confined to the fauces.

Albuminuria is frequently present from the earliest period. There is usually no dropsy. The diazo-reaction is seldom present.

Three forms of diphtheria may be distinguished according to their severity :—

1. *Benign* form. The general symptoms are very mild, and there are only a few scattered patches of membrane on the tonsils. Patients may be convalescent in a few days. No doctor may be consulted, and hence they are very likely to spread infection.

2. The ordinary form just described. If the membrane does not spread, a gradual convalescence may set in about the fourteenth day; or death may result from asthenia or cardiac failure. Laryngeal diphtheria leads to death from asphyxia.

3. The *malignant* form. Symptoms are severe from the start; there is great prostration, the typhoid state develops early, and there is a marked tendency to hæmorrhages. Death takes place in a few days.

Complications, beside those described, are—

1. Formation of membrane on external wounds.
2. Severe ulceration of the throat.
3. Cardiac failure, and rarely ulcerative endocarditis.
4. Thrombosis of veins.
5. Pulmonary affections (bronchitis, collapse, broncho-pneumonia).
6. Nephritis.
7. Meningitis.
8. Otitis media.
9. Conjunctival diphtheria (rare).

The most important sequela is *post-diphtheritic paralysis*. This is a peripheral neuritis, and may vary much in its distribution and severity—usually coming on within three weeks of apparent recovery. The more constant symptoms are—

1. Anæsthesia and paralysis of soft palate.
2. Loss of accommodation, with squint or diplopia.
3. Loss of deep reflexes.
4. More or less wasting of paralysed muscles, with reaction of degeneration.

The paralysis may be much more extensive, and if the intercostal muscles or vagi become affected, then the prognosis is very unfavourable. Usually, however, the paralysis passes off in time under suitable treatment.

Special Points.—Note—

1. The tendency for serous membranes to be involved in the course of septicæmia due to “mixed infection” (streptococcus).
2. The marked depression.
3. The red areolæ around the exudation.

Remember the membrane may be very localised, and not visible without a post-nasal or laryngoscopic examination.

Diagnosis is easy in well-marked cases. Wherever there is doubt, a bacteriological examination of the exudate, removed on a sterilised swab, should be at once undertaken. The swab is rubbed over the surface of blood-serum in a culture-tube, and is ready for examination after incubation for twelve hours at 37° C. In doubtful cases antitoxin should be given without waiting for this confirmatory evidence.

Treatment.—

1. Isolation must be continued for at least three weeks after the throat is clear, or till all discharge from the throat, mouth, or ears has ceased.

2. The *antitoxic serum* has greatly reduced the mortality from diphtheria. The treatment should be begun early, and the dose must vary with the period of the disease and the

gravity of the case. From 2000 to 8000 units (10 to 40 cc.) should be at once injected, another dose of 2000 to 4000 given in twelve hours in urgent cases, and a third twenty-four hours afterwards. Much larger doses may be given if the case is seen for the first time in a late stage. The age of the patient does not affect the dose. The membrane usually begins to separate in from twenty-four to thirty-six hours. Strict antiseptic precautions must be observed.

As the serum of an immunised animal not only has an important curative influence in cases of diphtheria, but also protects "contacts" against infection, it must also be used for prophylaxis. For this purpose a dose of 300 units may be given to a child, 500 to an adult. "The antitoxin unit is the amount of antitoxin which, injected into a guinea-pig of 250 grammes in weight, neutralises 100 times the minimum fatal dose of toxin of standard weight and strength."

3. Locally, frequent syringing of the fauces and nasal fossæ with astringent antiseptic solutions, such as chlorate of potash and hydrochloric acid, or formalin (1 in 200). In the early stages of laryngeal cases, hot sponges to the larynx, and the bronchitis kettle. Tracheotomy must be resorted to when the larynx is seriously involved.

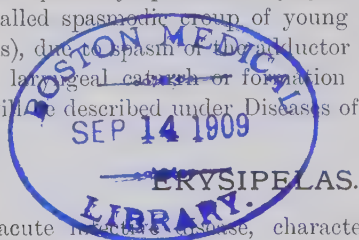
4. Avoid all lowering measures, and treat symptoms on general principles. Special attention must be paid to the condition of the heart.

The "post-diphtheritic" paralysis must be treated with careful hygiene, strychnine, massage, the interrupted current if necessary, and nourishing diet.

CROUP.

It is now universally recognised that most of the cases which used to be known as "membranous croup" are really of diphtheritic origin, and that the "croupy" symptoms are due to the existence of a primary laryngeal diphtheria. False membrane may also be formed in the larynx in the course of other diseases of bacterial origin, such as scarlet fever, small-

pox, measles, and whooping-cough; or it may arise as the result of injury from scalds, etc. In such cases the "croup" is merely a complication of the causative disease. The only independent maladies which can be called "croup" are non-contagious. Croupy symptoms may be due to an acute catarrhal laryngitis precisely similar to that of adults, no false membrane being formed. But since it occurs in young children, in whom the larynx is very narrow, the swelling of the mucosa causes the special symptoms of laryngeal stenosis. There is also the so-called spasmodic croup of young children (laryngismus stridulus), due to spasm of the adductor muscles of the larynx, without laryngeal catarrh or formation of membrane. These forms will be described under Diseases of the Larynx.



An acute infectious disease, characterised by a spreading inflammation of the skin and general febrile disturbance, and due to the presence of the *Streptococcus erysipelatis*.

Ætiology.—Erysipelas is widely distributed, but most common in temperate climates. It may be epidemic, but is usually endemic. It is commonest in springtime. Dirt and insanitary conditions favour its development. Important predisposing causes are chronic alcoholism, chronic Bright's disease, recent delivery, and the existence of wounds or abrasions. The virus clings to rooms and furniture, and can be conveyed by a third person, but is not active at any great distance.

The micro-organism is the streptococcus of Fehleisen, which grows in long chains. It may be cultivated outside the body, and stains with the ordinary aniline dyes. It is found in the lymphatics at the advancing border of the rash.

The incubation period is from two to seven days.

Morbid Anatomy, as regards the skin, is that of simple inflammation, in which the related lymphatic glands take part. There may be visceral complications of a septic nature, as

infarctions of lung, spleen, or kidney ; malignant endocarditis ; pericarditis or pleurisy ; and rarely meningitis. There is moderate leucocytosis.

Symptoms.—In a typical case of erysipelas of the head and face the onset is abrupt, and often attended with rigor or vomiting. The temperature rises sharply—it may be to 104° or even higher. Very soon a sharp red patch appears on the skin, either where there is a wound, or at the junction of skin and mucous membrane (inner canthus, angle of mouth, etc.). This rapidly spreads in all directions, the centre at the same time becoming paler. The edges are raised and hard, the surface red, tense, and painful, pitting on pressure. Superficial blebs appear on the surface, their contents being hæmorrhagic in bad cases. The face is enormously swollen, and the eyes are often closed. The cervical glands are swollen and tender. Mucous membranes may also be affected. The mouth may be attacked by extension from the face, and the larynx, pharynx, or nose may be involved. After a few days the inflammation ceases to spread, desquamation takes place, and defervescence occurs by crisis.

Constitutional disturbance is usually slight, but there may be delirium at night even in mild cases. In chronic alcoholics, and in the elderly, the prognosis is less favourable. The typhoid state is prone to develop, and death from toxæmia to result.

Occasionally the inflammation, while healing on the face, spreads to the trunk, and thence in similar fashion to the limbs (*erysipelas migrans*).

Complications (besides those already mentioned).—

1. Local suppuration, sometimes leading to
2. Septicæmia.
3. Extension to the larynx ; danger of death from asphyxia.
4. *Albuminuria* is very common, though nephritis is rare.
5. Pneumonia (rare).

Treatment.—In most cases little is needed beyond isolation and careful nursing. The strength should be maintained by

appropriate diet, and alcohol given when indicated, but not as a routine practice. Sedatives may be given for the delirium, or if this is violent, morphia or hyoscine. Cold sponging is to be preferred to antipyretics.

Internally, large doses (5ss-ʒi every four hours) of Tr. Fer. Perchlor. are often recommended, but there is really no specific remedy. Antistreptococcic serum is not an unqualified success. Locally, applications should be used such as will protect the parts from air and relieve pain. For this purpose one may paint with flexile collodion, apply a dusting powder, and wrap in cotton wool; or hot carbolie fomentations (1 in 40) may be used. Various applications are advised to prevent the spread of inflammation, the best means being the injection of antiseptic solutions beyond the spreading edge.

THE SEPTIC DISEASES.

Under this heading we mean the clinical phenomena attending the introduction into the blood of living micro-organisms or their products. The organisms mainly concerned are those of suppuration (*staphylococcus* and *streptococcus pyogenes*). There are three types of septic disease:—

1. *Supremia*—i.e. the introduction of bacterial products (toxins) which *cannot* multiply in the living tissues; the symptoms following being in proportion to the size of dose introduced.

2. *Septicemia*—The introduction of living organisms which have the power of *living and multiplying in the blood*, but which do not give rise to the formation of metastatic abscesses. The symptoms are produced by the toxins which they elaborate *in the blood*, and therefore *bear no relation* to the amount of bacteria introduced.

3. *Pyemia* is a condition in which by the dissemination from the original focus of septic matter throughout the circulation, secondary foci of inflammation are formed in the various organs and tissues, these foci developing into metastatic abscesses.

No fixed line can be drawn between the three conditions. Sapræmia and septicæmia, for instance, are closely similar in their symptoms, the latter, however, being usually of greater gravity. They not infrequently coexist.

It should be remembered that the local source of infection is often obscure, and sometimes undiscoverable even after death ("cryptogenetic septicæmia").

The paths of secondary infection are as follows (Muir and Ritchie):—

1. By lymphatics, with extension to lymphatic glands and serous spaces related to the primary lesion.
2. By natural channels, such as the ureters and bile-ducts (infection of kidneys and liver).
3. By the blood-vessels: (a) by a few organisms entering the blood from a local lesion and settling in a favourable nidus; (b) by septic phlebitis with suppurative softening of the thrombus and resulting septic emboli; (c) by direct extension along a vein (spreading thrombosis).

Symptoms.—*Sapræmia* is generally due to absorption of products of putrefaction, such as decomposed placental remains, blood clots, etc.; to post-mortem wounds, or absorption of the microscopic layer of dead tissues killed in operations, even under antiseptic precautions. The onset is rapid. There are local pain and swelling in the wound, chilliness or rigor, the general symptoms of fever, and marked prostration. If not relieved by treatment, diarrhœa and emaciation follow, the typhoid state appears, and the patient dies comatose or from exhaustion.

In *septicæmia* the symptoms are similar, but more severe. The disease begins within three days of wound infection, with one or more rigors, and with high fever, which shows daily remissions or intermissions. The pulse is rapid, soft, and compressible. Prostration is very great. Jaundice and diarrhœa are common. There may or may not be delirium. Death may occur on the second or third day; if later, the typhoid state appears, and leads to death within a week or ten days. Mild cases may run a more chronic course.

Pyæmia arises in connection with infected wounds, or diseases due to the pyogenic organisms (gonorrhœa, empyema, puerperal conditions, etc.). The situation of the abscesses depends on the course of the circulation from the seat of infection. The symptoms are repeated rigors, high fever, remittent or intermittent, profuse sweating, great exhaustion, marked emaciation. Jaundice, vomiting, and diarrhœa are frequent. There are local signs of abscess, visceral or sub-cutaneous. Pleurisy, pericarditis, and broncho-pneumonia are frequent complications. In acute cases the typhoid state soon makes its appearance, and death takes place in about ten days. In more chronic cases visceral abscesses are not common, but there may be suppuration in the joints or muscles. Some of these recover, others die of exhaustion after a long and irregular fever.

Treatment.—In *sapremia* the treatment obviously is to stop the *local manufacture* of the poison by careful cleansing and rest (free drainage of wounds, washing out uterus, etc.); remember also a septic condition may begin as a *sapremia*, and end as a true *septicæmia*, through the local condition of the first state acting as a favourable nidus for the production of the second condition.

Septicæmia being due to the introduction of *living* organisms, all we can do is to—

1. Stop the entrance of *more* organisms by using the utmost antiseptic precautions.
2. Support our patient, till the fight between bacteria and tissues is ended, by stimulants and *quinine*.
3. Avoid all depressing antipyretics.

Antiseptics internally are advised by some, but it is very doubtful if we can kill those organisms already in the blood. Where the infection is demonstrably due to the streptococcus, anti-streptococcus serum may be tried. It has proved useful in some cases.

Pyæmia, as regards treatment, requires the same as advised under Septicæmia, with the addition that accessible abscesses must be opened and surgically cleansed.

In all cases, any discoverable primary focus of infection must be dealt with surgically.

PLAGUE—PESTIS.

A specific infectious disease characterised by great virulence and rapid course, accompanied by buboes or pulmonary inflammation, and due to the presence in the blood and tissues of *bacillus pestis*.

Ætiology.—The disease is liable to become epidemic in the presence of insanitary conditions, filth, and overcrowding. It is now very rare in Europe, the last small epidemic occurring in Glasgow in 1900. It is commonest in warm weather. The contagion attaches itself particularly to houses and to fomites, but it is by no means so virulent as that of small-pox or scarlet fever. Previous to an outbreak, the disease usually appears in rats and mice, which die of it in large numbers. It spreads among them through the fleas with which they are infested.

The micro-organism is a minute bacillus, thick, and with rounded ends, which stain more deeply than the central portion. It grows on ordinary media, and stains with ordinary dyes.

The average *incubation period* is from three to five days.

Morbid Anatomy.—The most characteristic appearances are those of bubo, and of widespread hæmorrhages into all the organs and tissues. Degenerative changes are present in the heart, liver, spleen, and kidneys. The buboes consist of swollen and congested lymphatic glands embedded in a hæmorrhagic exudation into the adjoining areolar tissue. Later the glandular substance breaks down and becomes purulent. In the pulmonary form scattered pneumonic patches are to be found; and the spleen is enlarged, and may show infarctions.

Symptoms.—There may or may not be slight premonitory symptoms, lasting one or two days. The invasion is usually sudden, attended with high fever, vomiting, headache, suffusion of the eyes, and sometimes with rigor. There is great lassitude, the face has an anxious or a dazed expression, and the gait is staggering. Hearing is dulled, and the speech is thick and indistinct. The tongue is swollen, furred, and dry. There may be diarrhœa or constipation. Delirium ensues, the typhoid state sets in, and death may result in a few days.

In about 78 per cent of the cases (*bubonic plague*), buboes appear between the second and fifth days. They are commonest in the groin, less common in the axilla, and least common in the angle of the jaw. They are usually single, large, and very tender. The skin over them is inflamed.

In favourable cases convalescence occurs from the sixth to the tenth day, but the bubo continues to enlarge, breaks down, and is discharged in the form of pus and sloughs, which may persist for weeks.

In another form of the disease (*pneumonic plague*) there are no buboes. High fever and severe prostration are present, along with cough, and a profuse, watery, blood-stained (but not rusty) spit. Moist râles at the bases, and scattered pneumonic patches, are to be found, but the physical signs are not proportionate to the severity of the symptoms. The spit is often almost a pure culture of the bacillus. The mortality is very high.

Exceptionally severe and exceptionally mild forms (*septic plague* and *ambulatory plague*) are also to be met with.

The mortality varies in different epidemics from 50 per cent to 95 per cent, but appears to be less among Europeans.

In the recent Glasgow epidemic, of 28 cases recognised during life 28·5 per cent died. There were 8 others, inferentially plague cases, making 36 in all. The total deaths were 16, or 44·5 per cent. All the cases noted as pneumonic, and all those noted as septicæmic or diarrhœal, ended in death. Only one purely bubonic case proved fatal, and even that is noted as being “probably ultimately septicæmic.” Many of

the bubonic cases were, however, of a very mild type (pestis ambulans).

Only the pneumonic and septicæmic forms gave rise to secondary cases; the bubonic did not. In the former cases the bacilli were disseminated in the sputum or alvine discharges; in the latter locked up in the glands.

Special Points—

1. Rapidity of onset and course.
2. Extreme prostration.
3. Presence of characteristic bubo.
4. In the pneumonic form, diagnosis mainly depends upon bacteriological examination.

Prophylaxis.—Rigorous isolation must be carried out, and continued for a month after recovery. All excreta must be disinfected, and also clothes and utensils. Attendants and relatives, where the disease is epidemic, should be inoculated with Haffkine's prophylactic (a culture grown on broth for six weeks, and then brought for one hour to 70° C. Of this 2 to 5 cc. are injected. Smart febrile reaction follows, and lasts about two days.) Rats should as far as possible be exterminated, and their bodies burnt.

Treatment consists mainly in nursing and in supporting the strength. Ice-bags to the head, cold sponging, avoidance of depressant antipyretics, stimulation by alcohol and ammonia, are the main indications. Locally, pain in the buboes may be relieved by ice-bags or morphia. They should be opened when pus has formed.

Serum-therapy was introduced by Yersin, but the results in different epidemics have proved very conflicting. Where the mortality is so great, any method at all promising may well be tried, and serum-therapy is based upon the rational principle of neutralisation of the toxin. The best results appear to be obtained when the serum is given intravenously, and in large doses.

CHOLERA.

A specific infectious disease, occurring in epidemic form, and characterised by violent purging and vomiting, pain, cramps in the legs, suppression of urine, and intense collapse.

The mouth of the Ganges is claimed as the home of cholera, and although epidemics have occurred in most parts of the world, they can always be traced back to India.

Ætiology.—The disease is not directly contagious. Contagion is conveyed by stools, contaminated water, and vegetables or other food-stuffs washed in it. It breaks out principally in summer and autumn, and attacks all ages and both sexes alike. It spreads along trade routes no faster than the rate of human travel.

The Germ is a spirochæta, known as the comma bacillus of Koch. It is about half the size of, but thicker than, the tubercle bacillus, and is curved, but may be spiral or shaped like an S. It is found in the dejecta and intestines of all patients affected. It does not occur in the internal organs or blood. The constitutional symptoms are therefore supposed to be due to the absorption of toxins from the intestine.

It grows in many media, but requires either a neutral or a slightly alkaline soil to flourish. It dies in sterilised distilled water; lives, but does not multiply, in sterilised drinking water; and multiplies rapidly in water containing dead vegetation.

Pettenkofer holds that “germs” develop in the subsoil during summer, and rise into the air as a “miasm.”

Pathology.—That of a severe inflammation of the mucous membrane of the small intestines, the process being specially marked around Peyer’s patches and solitary glands; usually there is no ulceration, although there is marked hyperæmia, and the intestine may appear thinned. It is filled with a turbid fluid, in which the bacilli are present in large numbers.

The blood is dark and thick, sometimes almost tarry through the drain of water from the system. The other organs show changes similar to those occurring in other virulent fevers. The tissues are dry, and the serous membranes dry and sticky. Note, however, that there *is rapid rigor mortis*, and sometimes *post-mortem rise of temperature*.

Symptoms.—After a short incubative period (from two to five days) a preliminary diarrhœa sets in, with more or less headache, vertigo, and nausea; then the characteristic diarrhœa commences. Most clinicians divide the disease into three stages—

1. Evacuative.
2. Algid or collapse.
3. Reaction.

The preliminary diarrhœa may also be reckoned as the *stage of invasion*, in which case four stages are enumerated.

Evacuative Stage.—Violent diarrhœa accompanied with intense pain, and cramps in calves of legs. The stools are at first fæcal, but soon become like “rice water.” The reaction is neutral or alkaline, the specific gravity 1006 to 1012; they show on standing—

1. Epithelial debris.
2. Threads of algæ.
3. Bacteria.
4. NaCl and triple phosphates.
5. Blood pigment and albumin.

Often the evacuations, which are extremely copious, are quite painless. Then vomiting takes place, first of food, but finally of matter similar to the stools. It may be almost incessant. There are great prostration, extreme thirst, and violent cramps. The external temperature is subnormal, and the pulse rapid and weak. Then the second or

Algid Stage commences. The collapse becomes extreme; the features are shrunken, livid, or ashy grey; eyeballs sunk in; the skin is shrivelled and wrinkled, and covered with a cold,

clammy sweat. Though the surface temperature is subnormal, the temperature rises in the rectum to 102° or more. The voice is husky ; the pulse is small and flickering, and often absent at the wrist. The purging usually ceases, but the vomiting continues. There may be a complete suppression of urine, coma, and death within a few hours ; or the patient may pass into the third, or

Reaction Stage.—The temperature gradually rises, and a red glow replaces the ashy appearance. Erythema and urticaria are frequent. The patient gradually recovers, the urinary secretion returning, or the improvement is checked by the onset of—

1. The typhoid state.
2. Inflammatory complications (pneumonia, enteritis, etc.).
3. Recurrence of severe diarrhœa.
4. Uræmia, coma, and death.

The stage of evacuation may last from two to sixteen hours, that of collapse no more than twenty-four. The duration of the reactive stage is less definite.

The mortality from cholera is greatest at the beginning of an epidemic. Very virulent cases may die before diarrhœa has begun (*cholera sicca*). Pregnant women almost always miscarry.

Treatment.—In the stages of invasion and evacuation diarrhœa should be checked, if possible, by Pil. Plumbi cum Opio, lead acetate, mineral acids, or morphia hypodermically. For vomiting, mustard should be applied to the abdomen, and ice given to suck. Cramps are to be relieved by friction and morphia. No food or stimulant should be allowed, and the thirst must be relieved by ice alone. In the collapse stage, warmth must be applied to the limbs, and if there is no purging, enemata of beef-tea and brandy may be given. Intravenous injection of warm saline solution is often at least temporarily successful (common salt gr. lx, carbonate of soda gr. xxx, warm sterilised water Oij), but equally good results are got by intracellular

injection, which is both safer and easier to carry out. In the stage of reaction, food must be given often, and at first very sparingly. Excessive fever should be checked by sponging, and continued suppression of urine by dry cupping and fomentations over the loins.

Prophylaxis.—The utmost sanitary vigilance, isolation, and quarantine; all soiled linen, etc., should be burnt; drinking-water to be first boiled, and avoidance of all irritating food. During cholera epidemics all cases of diarrhœa should be checked as rapidly as possible, by opium, chlorodyne, or other astringents.

Preventive inoculation has been introduced by Haffkine, who injects subcutaneously first a weak and then a strong vaccine, prepared from cultures of the cholera vibrio. The results appear to be distinctly favourable in diminishing liability to the disease.

DYSENTERY.

A term employed to designate various forms of intestinal flux, all characterised by the frequent passage of mucoid or hæmorrhagic stools, tormina, and tenesmus; and pathologically by inflammation, and sometimes ulceration of the large intestine. *Ætiologically* the various forms are probably distinct.

Ætiology.—Endemic and epidemic in tropical climates, where the epidemics are sometimes extremely fatal. In the temperate regions the epidemics are less severe, and occur mainly in armies, in times of famine, and in large public institutions (jails and asylums) where sanitation is defective. Sporadic cases are more common. Battlefields, dried-up river-beds, decayed vegetation, and dung-heaps form suitable soil for the poison, and a fitting nidus within the body is sometimes furnished by the ingestion of unripe fruit.

The contagion probably spreads through the stools, as in typhoid.

Specific Organisms.—Various organisms are described in connection with the different forms of dysentery. The best known is the *amœba dysentericæ*, found by Kartulis and others, in the stools, intestines, and in the liver abscesses of dysenteric patients.

Amœba is a unicellular organism, mobile, and shows—

1. Clear outer zone = ectosarc.
2. Granular inner zone = endosarc, containing vacuoles and nuclei.

But many authorities regard the amœba as an “epiphenomenon”—not specific, but implanted on a soil rendered suitable by the dysenteric process. It does not occur in all cases. Ogata has described a specific bacillus in the dysentery of Japan, and Flexner has found the same organism in the Philippines. There are also forms of dysentery in which neither organism is found.

The *incubation period* varies from three to eight days (Liebermeister).

Varieties.

1. Acute.
 - (1) Catarrhal: ending in grave cases in gangrene.
 - (2) Amœbic or tropical.
 - (3) Diphtheritic, divided into primary and secondary.
2. Chronic.

The **Morbid Anatomy**, obviously, will vary with the type of disease; but after all, the essential *lesions differ only in degree*. As a typical example, take—

1. *Catarrhal Form.* Site—lower part of ileum and large intestine. Here we have to do with a severe inflammation of a particular mucous membrane; therefore we get—

- (1) Hyperæmia.
- (2) Exudation rapidly becoming purulent.
- (3) *Necrosis of exposed follicles.*
- (4) *Separation of sloughs and formation of dysenteric ulcer.*

Put into other words, the inflammatory process is very severe, and only differs from inflammation of any other mucous membrane *because of the distinctive characters possessed by the gut involved.*

2. *Amœbic Variety.*—*Ulceration is more marked.* The whole of the large gut may be riddled with ulcers, and abscess of the liver much more frequently results. These abscesses may be large and single, or small, superficial, and multiple. Perforation into the right lung sometimes occurs.

3. *Diphtheritic Type.*—In the *Primary form* the necrosis is extremely rapid. It may affect only the superficial layers of the mucosa, or the whole mucosa may form “a black, rotten, friable, coloured mass” (Rokitansky).

The Secondary type is peculiar as being associated with pneumonia, chronic Bright’s disease, or cardiac disease. *The exudation is often pseudo-membranous*, lying upon, not within, the mucosa. This condition *often forms the last stage of the diseases mentioned.*

Characters of the Dysenteric Ulcer.

1. Irregular undermined edges.
2. Base may be formed of any of the coats.
3. If they heal they cause much contraction.
4. Usually seen best marked on ridges of large intestine.

Symptoms of acute dysentery vary with the type of disease, but one tends to merge into another. The onset may be sudden, or there may be previous diarrhœa. There are frequent or incessant calls to stool, with pain, griping (tormina), and constant sense of weight in the rectum (tencsmus). The stools are *very small*, and composed of slimy mucus, which soon becomes blood-stained. Passage of a stool gives no relief; straining continues, and in grave cases from 50 up to even 200 stools may be passed in twenty-four hours. The patient loses strength. There are slight or moderate fever, great thirst, dirty tongue, dizziness, and dry skin. In the graver forms he may die of exhaustion, or the general condition may rapidly

assume a low and typhoid state, or death may result from pyæmia or perforation. In milder cases the disease subsides rapidly or slowly, or it may lead to chronic dysentery. Where there is ulceration, the stools contain clumps of grey or fœtid sloughs, and are very offensive; where there is gangrene, the sloughs are larger (there may be long tubular structures composed of mucus), the fluid portion is blackish, and the odour intensely disgusting. Pure blood may be passed as the result of ulceration; and in the late stages almost pure pus. The "frog's spawn" or "boiled sago" stool is commoner in chronic dysentery.

Complications.—As the blood from the intestines is returned by way of the liver, we naturally may expect trouble from products so carried, and usually we get—

1. Single or tropical abscess of liver, or (much less common)
2. Multiple pyæmic abscesses. Other complications are—
 - (1) Hæmorrhages and perforation.
 - (2) Peritonitis or pneumonia, etc.

Chronic Dysentery may be chronic from the beginning, or a result of an acute attack through persistent suppuration of the submucous abscesses.

Pathology.—

1. There are chronic ulcers with thickened edges, and areas of scar tissue.
2. The deep coats of the gut are much thickened and contracted, and sinuses are common.
3. The calibre of the bowel is much diminished from contraction, and in parts dilated from adhesions.

Symptoms.—Slightly bloody, painful diarrhœa, much mucus being mixed with the stools; the appetite is absent; tongue glazed and red; intense anæmia; and patient has a shrunk look. The emaciation may be extreme, *but the spleen is not usually enlarged.*

Treatment.—

Acute.—Rest in bed, fluid food (milk, chicken-tea, barley-water, etc.). Morphia and small laudanum enemata for pain or tenesmus. After keeping stomach empty for three hours, m xx of laudanum, then gr. xx-xxx of ipecacuan, followed by *absolute rest*. If vomited, repeat the dose. Same treatment every 8 hours or so for two or three days. Or give Sodii Sulph. ʒj every two hours till feculent motion produced, and then so as to produce two or three motions daily. When mucus and blood have gone, bismuth and opium till motions are solid.

Calomel gr. $\frac{1}{4}-\frac{1}{2}$ every hour, or minute doses of perchloride may also be given in the acute stage.

Chronic.—Careful diet; plenty of fresh air; rest in bed; salol, quinine, bismuth, and Dover's powder; injections of silver nitrate, 10 grs. to 20 oz. of water; inject two pints. Later, arsenic and hypophosphites.

YELLOW FEVER.

An acute specific fever of limited geographical distribution, characterised by jaundice, albuminuria, and tendency to hæmorrhages, particularly from the stomach; and pathologically by fatty degeneration of the liver and acute nephritis.

Ætiology.—The disease is endemic in the West Indies, on the coasts of Central and South America, and on the West African coast. From these centres it spreads in epidemic form, usually by means of shipping, to other and sometimes remote regions, where, however, it has never taken a firm hold. It is most severe at low levels, and in crowded towns upon the coast-line, and is especially favoured by warmth and moisture. There is no racial immunity, although negroes in the endemic areas suffer less severely and less frequently than whites. One attack confers almost complete immunity, *so long as the subject remains in the infected area* (repeated inoculation?).

After introduction of a case to a new area, others do not appear for from two to three weeks. The *individual* incubation period, after inoculation, is from three to six days.

The disease is not transmitted by direct contagion. It is said to be spread by fomites, but Osler cites recent experiments disproving this. It is now proved that it may be spread by the bites of mosquitoes (*stegomyia fasciata*, and probably other species) which have previously bitten infected persons. From ten to twelve days are required for incubation in the mosquito before its bite transmits infection.

The specific organism is probably bacterial (Durham), but has not yet been discovered, although Sanarelli has described a *bacillus icteroides* in connection with the disease.

Morbid Anatomy.—The skin is jaundiced. The most important changes are in—

1. *Liver*.—Size about the normal, colour pale yellow with hæmorrhagic patches; cells atrophied, necrotic, and showing *marked fatty degeneration*.
2. *Kidneys*.—In a state of acute nephritis, and much engorged; cells full of fat globules.
3. *Stomach*.—Injected and ecchymosed, coated internally with altered blood; contains “black vomit.”

Symptoms.—The onset is usually sudden, accompanied by chilliness or rigor, severe headache, and pains in the back or limbs. The temperature runs up to 101°-104°, and sometimes higher. The face is bloated and flushed, the conjunctivæ injected and bright red; there may be a slightly jaundiced tint. The tongue is pointed, red at the tip and edges, and furred in the middle. There may be simple vomiting. Albuminuria may be present from the first day. The pulse is full and strong, but *slow relatively to the temperature*, and *tends day by day to become slower while the temperature keeps high*. It may fall even below the normal. In mild cases convalescence may begin about the third day; in the severer forms the case goes on to

The *second stage*, which commences about the fourth day. Its most prominent features are—

1. “Black vomit.” The vomit is at first watery, but becomes more and more mixed with altered blood, and like coffee-grounds in appearance.

2. Other hæmorrhages, as epistaxis, hæmorrhage from the bowel, etc. Metrorrhagia is common, and pregnant women always abort.
3. Highly albuminous and scanty urine. Complete suppression is not uncommon, and if it persists for twenty-four hours the case almost always ends fatally.
4. Jaundice, beginning in the conjunctivæ. The tint is oftenest a faint lemon yellow, but it may be deeper, or may even be a dark orange brown.

There are also asthenic symptoms—intense collapse, shrunken features, etc.; or the typhoid state may occur.

The mortality is very variable in different epidemics and races. "It ranges anywhere from 5 to 75 per cent of those attacked" (MANSON).

Treatment.—It is well to begin with a smart purge (calomel or castor oil); afterwards laxatives must be used cautiously. Nothing but small quantities of water or iced water need be given in the first stage; large quantities will be vomited. Stimulants are seldom required so early. They may be used later in the presence of collapse or asthenia, and food may then be given by the rectum. The headache may be relieved by an ice-cap, and sponging may relieve the fever. Drugs are of little use, though astringents may be tried for hæmorrhage.

Prophylactic.—In general, the breeding-places of mosquitoes should be destroyed as in malarial regions, and cisterns protected by wire-gauze. Adult insects should be killed, especially in infected houses. During the fever season the nights should be spent away from the town in a healthy suburb, preferably at some height above the coast-line.

MALARIAL DISEASE.

A specific infective disease caused by the presence in the blood of the *plasmodium malariae*, and characterised by (1) periodically recurring paroxysms of intermittent fever; (2) *continued* fever with well-marked *remissions*; (3) certain per-

nicious, rapidly fatal forms; and (4) a chronic cachexia with anæmia and enlarged spleen.

Ætiology.—The disease has its headquarters in tropical and subtropical regions, but occurs also in less severe forms in more temperate climates, in which the period of fresh infection is the summer and autumn. Warmth, abundant vegetation, stagnant surface water, and hence low-lying and marshy districts, favour its development, while the opposite conditions retard it.

It is spread by the bites of mosquitoes of the genus *Anopheles*, in the body of which the parasite completes the extra-corporeal part of its life-history.

The malarial parasite is a protozoon of the order Sporozoa, and belongs to the group of the Hæmamœbidæ. The term *plasmodium* is applied to it only as it exists in human blood. Its life-history is as follows:—In its youngest form it is a small amœboid body (*amœbula*) occupying the interior of a red blood corpuscle, and consisting of nucleus, nucleolus, and surrounding protoplasm. The amœbulæ rapidly grow and become pigmented by converting the corpuscular hæmoglobin into melanin. They reach maturity in 24, 48, or 72 hours, according to the species. When mature some of them become *sporocytes* (asexual forms), and others *gametocytes* (male and female sexual forms). The sporocytes then form a “rosette”-shaped body, consisting of a variable number of nucleated segments or spores, which rupture the containing corpuscle, and pass with the liberated melanin into the liquor sanguinis. The melanin is absorbed by the phagocytes, and carried to the central organs; while the spores attack fresh corpuscles, and repeat the same cycle of development.

The sexual forms, or gametocytes, do not divide in the body, but circulate unchanged. When they are drawn into the stomach of the mosquito, they burst the containing corpuscle. The male gametocyte puts forth four or more actively mobile flagella (microgametes), which are really spermatozoa. These detach themselves from the parent cell,

and attack the female gametocytes (macrogametes). One of them succeeds in penetrating and fertilising a macrogamete, which now becomes elongated and of the shape of a spear-head (zygote). The mobile zygote penetrates the stomach wall, fixes itself on the outer surface of the stomach, and develops there, by segmentation of the nucleus, into a capsule filled with numerous delicate spindle-shaped *blasts*. This process occupies at least a week. The capsule ruptures, and the blasts are carried by the circulation to the salivary gland and thence to the salivary duct. They are then injected by the mosquito into the next puncture that it makes, and set up a fresh malarial infection in a new host.

Three forms of the parasite occur in man, all with the same life-history, but with individual differences:—

1. The *quartan* parasite occupies medium-sized corpuscles. Its amoeboid movement is sluggish. It produces about 8 spores, and its asexual life-cycle lasts 72 hours. The gametocytes resemble the sporocytes before spore-formation.
2. The *tertian* parasite occupies large pale corpuscles. The movements are active. It produces 15-20 spores, and its life-cycle lasts 48 hours. The gametocytes resemble the sporocytes.
3. The *estivo-autumnal* parasite occupies medium-sized corpuscles. The movements are at first active. It produces numerous spores. The life-cycle lasts 24 hours (quotidian form), or 48 hours (tertian form). The gametocytes are crescentic, with pigment granules in the centre. Only gametocytes and young amoebulae are found in the peripheral blood.

The liberation of each swarm of spores coincides with an attack of fever. Thus with simple tertian or simple quartan infection a paroxysm occurs every second or every third day. But in tertian ague there may be two broods of spores coming to maturity on alternate days, so that what appears to be a

quotidian ague is really a double tertian ; and in quartan ague there may be two broods, with fever on two successive days, and a day of interval, or three, with a paroxysm every day. In one form of æstivo-autumnal fever, as shown above, sporulation occurs every 24 hours, and the paroxysm is therefore quotidian.

Not only fever, but also great destruction of red blood corpuscles, follow sporulation. Both are probably due to the liberation at that moment of a toxin possessed of heat-producing and hæmolytic properties.

The *incubation period*, as determined experimentally, varies from 6 to 20 days.

Morbid Anatomy.—The principal changes are—

1. Enlargement of the spleen, sometimes very great. In recent cases the organ is soft ; in older cases firm (“ague-cake”).
2. Enlargement and congestion of the liver.
3. Congestion of bone-marrow, of cerebral vessels, and of kidneys.

In all these organs the vessels are full of plasmodia and of melanin. There is also present a yellow pigment, not confined to the vessels, which consists of altered hæmoglobin. Marked diminution of the number of red cells, and pigmentation of the leucocytes, are conspicuous features. The mononuclear leucocytes are also relatively increased for some time after the paroxysm.

In *malarial cachexia* the spleen is not necessarily pigmented. The enlargement is mainly due to thickening of the capsule and trabeculæ, and the organ is hard.

I. Intermittent Type (Ordinary Ague). The Paroxysm.—After some days of premonitory symptoms of more or less general malaise, produced by the multiplication of the parasites before they are numerous enough to determine an attack of fever, the typical attack comes on, and usually consists of three stages—cold, hot, and sweating stages.

Cold Stage.—Patient shivers violently, teeth chatter, skin is pale and blue, and papillæ are raised (goose-skin); there is a great tendency to collapse. There is often vomiting. The temperature, though much lowered externally, is, however, raised in the rectum. As may be expected with such an extreme contraction of the superficial capillaries, the urine is pale, copious, and of low specific gravity. This stage lasts from a few minutes to an hour or more. Towards the end of the stage the temperature may be 103° to 106° .

Hot Stage.—It may be gradual or sudden in its onset. The skin becomes hot and burning, and a patchy rash may form. There is vomiting or complete anorexia; throbbing of the carotids with intense headache, and there may be delirium. The temperature is often 106° or more, and the pulse is rapid and full. Often crops of herpes form at the mouth. This stage lasts from one to many hours, being longest in the æstivo-autumnal form. Enlargement of the spleen can often be demonstrated. Urine is scanty, of high specific gravity, and contains a large quantity of *urates and urea*. Albuminuria is frequent.

Sweating Stage.—The sweating commences at the roots of the hair, but soon becomes general and profuse. The pulse gets softer, the temperature falls gradually to the normal, and the patient is restored to the normal condition, the enlargement of the spleen diminishing or disappearing. During the sweating stage the urine is of high density, and scant in quantity; *urates* are more abundant than *urea*. The patient remains anæmic. This stage lasts from two to four hours. Attacks may recur daily, as has been seen, or every second or third day (quotidian, tertian, and quartan ague). The simple tertian and quartan paroxysms last ten to twelve hours, the æstivo-autumnal quotidian six to twelve, the æstivo-autumnal tertian twenty-four to forty.

Paroxysms are said to “anticipate” when they come on a little earlier each succeeding day, to “postpone” when they come on a little later.

II. Remittent Type (*bilious remittent fever*).—In this form the fever is more or less continuous, with well-marked remissions but not intermissions. The special symptoms are coated tongue, epigastric pain and tenderness, bilious vomiting, anorexia, constipation or diarrhœa, and jaundice. It tends to merge either into an ordinary intermittent attack or into a typhoid type of grave prognosis.

III. Pernicious Attacks are comparatively rare. They occur mainly in tropical countries, among those exposed to hardship or in whom resistance has been lowered by intemperance or previous attacks of malaria. They are sudden in onset, and of extreme gravity. The following are the chief forms:—

1. Hyperpyrexial, the temperature running up to 107° - 110° , or even higher.
2. Cerebral, due to plugging of various cerebral centres by plasmodial emboli. Comatose, convulsive, and paralytic forms are described.
3. Algid. There is no febrile reaction after the cold stage, and the case ends in collapse.
4. Choleraic and dysenteric. The choleraic stools of malaria always contain some bile.

First attacks of malarial fever, especially in the tropics, are usually either remittent or continued, and only gradually assume the intermittent type. In temperate climates they may be intermittent (and most frequently tertian) from the first.

IV. Blackwater Fever or Hæmoglobinuric Fever occurs especially in tropical Africa and more rarely in certain parts of India. Recurrence may take place after recovery even when the patient has been removed to temperate climates. It is uncommon among natives, and usually occurs after a prolonged residence in malarial regions, and repeated attacks of malaria, which have led to anæmia and debility. Its etiology is obscure, malarial parasites being absent or scanty in the blood during the attack, and in internal organs after death. Manson inclines "to regard the parasite . . . as being in some respects

different from that of ordinary malarial affections." Many observers, among them Koch, consider the prolonged use of quinine as the cause of the hæmoglobinuria, but there is much evidence against this view. One attack predisposes to another. Essentially the disease is an acute hæmolysis. Red corpuscles are but sparsely found in the urine, but hæmoglobin is present in abundance. There is cloudy swelling of the hepatic and renal cells. Numerous casts are found in the renal tubules. The skin and conjunctivæ are yellow, or more darkly brown from true jaundice.

The attack may commence as an ordinary malarial paroxysm, but after a few days there is a severe rigor, and the urine becomes very dark or even black. It is at first copious, but later diminished or suppressed. There is yellow discoloration of the skin and sclerotics, also bilious vomiting, and sharp pains in the loins and epigastrium, usually also bilious diarrhœa. The condition may pass off in a few hours, and not recur. More often there are recurrences with each attack of fever, or there may even be no remission at all. Marked anæmia and prostration develop, and death is very frequent.

While in the malarial zone, the slightest chill or overstrain may provoke recurrence. After the attack is arrested, therefore, the first aim of treatment is to remove the patient to a temperate climate, at a season of the year which is not too cold. Even so, recurrences may take place, and prove fatal; but the tendency generally dies out in about six months. Of drugs, some advocate quinine in large or moderate doses, frequently repeated; others, who consider quinine the cause, large doses of calomel. Tannin and other astringents, turpentine, etc., have been used. Fluids should be given often and freely, to flush the tubules and avert suppression. On no account must the patient sit up in bed, owing to the risk of cardiac failure.

V. Malarial Cachexia. — After repeated or prolonged attacks of malarial fever, and also in those who have been long

resident in malarial countries, a peculiar cachexia is apt to develop which is characterised by

1. Anæmia, often intense, the skin being of a sallow earthy colour.
2. Tendency to hæmorrhages—epistaxis, purpura, etc., and retinal hæmorrhages.
3. Irregular attacks of fever.
4. Great and sometimes enormous enlargement of the spleen ; to a less extent enlargement of the liver.

The parasites may be altogether absent in malarial cachexia.

Diagnosis of typical forms of ague is easy ; in atypical forms the response to quinine, and above all *the results of examination of the blood*, will establish the diagnosis. Those intending to practise in malarious regions should make themselves familiar with the various appearances of malarial blood.

Treatment of Malaria in general.—The specific for malarial fevers is quinine, which, properly given, destroys the parasites in the blood. It should be given promptly, and in the early stages in doses up to gr. xxx in the twenty-four hours, and continued in doses sufficient to produce moderate ringing in the ears for a week after the parasites have disappeared from the blood (Ross). The dose may then be gradually reduced, but the drug must be continued for at least three months. It may be given in capsule or in acid solution. In grave or pernicious cases it may have to be given hypodermically, to secure a prompt effect, in doses up to gr. x thrice daily. The injections should be made into muscle, not subcutaneously. In chronic cases small doses of quinine, if plasmodia are present, are enough. Arsenic, alone or with iron, is of great service, but iron alone has not so good an effect. Arsenic is useless in acute cases.

Prophylactically, 5 or 10 grains of quinine may be taken in the morning. Mosquito curtains, wire-gauze window screens, etc., must be employed ; in tropical towns Europeans should live in a separate quarter ; and a crusade against the larvæ of

Anopheles should be undertaken, by drainage, covering the surface of pools with a film of kerosene, and so forth.

ACTINOMYCOSIS.

A chronic infective disease due to the presence and multiplication of the "ray fungus" (*Actinomyces*).

Ætiology.—There is no evidence of direct infection from the flesh of diseased animals. The site of infection in man and animals is usually the mouth, and thus the fungus may be taken in with the food, or may find a lodgment in those who are in the habit of chewing straw, or who inhale the dust of grain in thrashing or chaff-cutting. It is common on various cereals.

General Characters (MACROSCOPIC)—

1. IN CATTLE.—The disease usually begins in the lower jaw, and causes sarcoma-like growths—bulky tumour-like masses—connected with the bone. The bone is softened and eroded, leading to the formation of an abscess. The tongue is also affected, hard indurated nodules being produced which give a gritty, woody feel; hence the name "woody tongue." The disease does not affect carnivora.

SITES of the lesion—

1. Jaws—tongue—neck—glands beneath the jaw.
2. Larynx—lung—alimentary tract.

2. IN MAN.—The disease also affects the jaw, and leads, as in cattle, to the formation of nodular masses, of deep-seated abscesses (retro-pharyngeal) in the region of the spine, etc.

Nodules also occur in the lungs—patches of chronic induration, abscesses, and cavities.

The intestines, liver, kidneys, and ovaries may also be affected.

The masses tend to suppurate, *not to caseate*.

Structure (MICROSCOPIC)—

The nodules are seen to consist of masses of granulation tissue. They have a sponge-like arrangement, and contain

small yellowish granules. They have a structure somewhat similar to that found in tubercle, being composed of—

1. The fungus—ray fungus—in the centre.
2. Round this there may be giant cells.
3. Epithelioid cells.
4. Small-celled infiltration and proliferation of connective tissue.

Mode of Inoculation—

1. Through the mouth by carious teeth, etc.
2. By the respiratory tract.
3. Through the intestinal tract.
4. Through the vaginal orifice (rare).
5. In many cases the mode of inoculation is unknown.

Ray Fungus—

1. *Characters.*—The exact botanical position of the ray fungus is not known. It is usually regarded as a streptothrix or cladothrix.

2. *Structure.*—The fungus consists of short threads or rods, often club-shaped, dotted, branched, sometimes calcified, arranged in a radiating manner round a common centre, composed of fine filamentous fibres. They form the globular masses already spoken of. The central part of the mass is the living cladothrix, the rays are *degenerated filaments*.

3. *Cultivation.*—Actinomycosis has been cultivated, but not with uniform success. It has also been inoculated, but here also, the results have not been altogether satisfactory.

Symptoms.—The symptoms of this remarkable disease depend entirely upon its mode of entrance, and the site of its most rapid multiplication; thus its pernicious influence may be most marked in—

1. The alimentary canal.
2. The pulmonary organs.
3. The brain.
4. The skin, etc.

Professor Grainger Stewart, in conjunction with Dr. Robert Muir, gives, in *Edinburgh Hospital Reports*, vol. i., a most exhaustive account of a case which began in the ovaries.

The *diagnosis* is dependent not so much upon the varying symptoms, as upon the recognition in the purulent discharges of minute sulphur-yellow masses, which under the microscope are found to consist of colonies of the fungus.

Treatment.—This is at present unsatisfactory, because the condition, in the first place, is not easily diagnosed; and secondly, when it is diagnosed in man, the extensive inflammatory changes, the numberless adhesions set up, etc., give little chance of the complete success of even surgical treatment. But where the disease is accessible, surgical removal is indicated. Even if only partial, this step, followed by free irrigation with antiseptics, is often of great value. Of drugs, potassium iodide (gr. xl- $\bar{5}$ i daily) is the most serviceable, and some cases have been cured by its use.

ANTHRAX.

Anthrax—also called charbon, malignant pustule, wool-sorter's disease, splenic fever, splenic apoplexy—is an acute specific disease which especially attacks cattle, horses, sheep, swine, and deer; but it also occurs in man, and is due to the agency of a specific bacillus—bacillus anthracis.

Ætiology.—Anthrax in animals is the result either of direct inoculation, as by fly-bites, or of grazing in infected pastures. In man it attacks those who have to do with the slaughtering of cattle, or the handling of carcasses or hides, as butchers, tanners, wool-sorters, and hair-combers. The poison may also be carried by flies.

The bacillus anthracis has the following characters:—

(1) *Shape.*—As it occurs in the blood of affected animals, bacillus anthracis consists of straight rods joined end to end, with blunt, slightly curved extremities. It is motionless—thus

differing from *bacillus subtilis*. It does not form spores in the blood, but does so in presence of free oxygen. Thus spores are to be found in the secretions of mouth, nose, and intestine, and also in shed blood.

(2) *Size*.—They vary considerably—from 5 to 10 or 20 μ . in length—1·2 μ . broad.

(3) *Cultivation*.—*Bacillus anthracis* will grow on gelatine, agar-agar, potatoes, in hay infusions, in aqueous humour. It liquefies gelatine, and sinks to the bottom of the vessel in whitish masses. It requires oxygen for its growth. When cultivated in potatoes, it gives rise to dry, creamy, yellow-coloured masses. When grown at a temperature of 24° to 42° C., it forms long filaments full of spores.

If a diseased carcase be not opened, spores are not formed, and the bacilli die in a short time; but if it be opened, and the blood and tissues exposed to air, spores are formed and the carcase therefore constitutes a lasting source of infection.

Division.—Anthrax, as it occurs in man and animals, may be—(1) Local; (2) General.

In man the disease manifests itself chiefly in the local form; in cattle, etc., the general form is more common.

Characters.—(1) In cattle; (2) In man.

1. ANTHRAX IN CATTLE—

1. There is but little local lesion. The chief changes are in the *spleen*, which becomes enormously swollen, and like a mass of blood-clot—hence the name, splenic apoplexy.

2. Hæmorrhages occur in such organs as the lungs, wall of heart, cortex of kidney, brain and its membranes.

3. There are areas of inflammatory exudation—cellulitis, effusions into serous cavities.

4. The blood has a dark colour, and is crowded with bacilli.

5. The lymphatic glands are also affected—especially round the pharynx, œsophagus, and stomach.

2. ANTHRAX IN MAN—

1. MODE OF INOCULATION—

- (1) By wounds.
- (2) By the respiratory tract.
- (3) By the alimentary tract.

2. DIVISION.—There are two forms in man—local and general. The local form is again subdivided into *malignant pustule* and *anthrax-œdema*, and the general into bronchial and intestinal forms.

(1) *Local Form*.—(a) *Malignant Pustule* is due to inoculation ; hence it occurs on exposed parts—on the face, neck, lips, hands, arms. It commences as a small papule, which becomes vesicular, and round which there is inflammatory induration. In about thirty-six hours the summit of the papule forms an eschar, round which there may be a ring of vesicles. There are great induration and œdema ; the lymphatics are inflamed, and the glands swollen. The temperature is at first high, but may afterwards be subnormal. Death may occur in three to five days, or slow recovery after sloughing out of the eschar. The bacillus is found in the superficial and subcutaneous lymphatics of the affected area, and afterwards in the blood, etc.

(b) *Anthrax-œdema*.—In this form eschar and induration are absent, the constitutional symptoms are very grave, and the swelling is of the nature of an extensive and spreading œdema, which may go on to gangrene. It is much more fatal than malignant pustule.

(2) *General Form*.—Rare in man, but may affect—

(a) *The Respiratory Tract*.—Wool-sorter's disease. The primary lesion is usually in the lower part of the trachea and larger bronchi, where there are patches of intense swelling of the mucous membrane, with hæmorrhages and ulcerations. There may also be broncho-pneumonic patches in the lungs. Great swelling of mediastinal glands, which, from hæmorrhages, look like blood-clots. In these cases there

are few bacilli in the blood, but they are found in great numbers in the bronchi and in lymphatic glands. The disease begins with chill or rigor, fever, headache, vomiting, or diarrhœa, and marked prostration. There are predominant pulmonary symptoms corresponding to the local lesions, hurried breathing, and cyanosis. Delirium is common, but the mind may be clear. Death occurs in three or four days. Survival for more than a week usually means recovery.

- (b) The *Gastro-intestinal form* gives rise to diffuse inflammation, with partial detachment of mucous membranes. Hæmorrhages also occur in this situation. The symptoms are those of intense poisoning—severe vomiting and diarrhœa, and possibly blood-stained stools.

Treatment.—In the local form, the swelling should be excised, if not too large, or crucial incisions should be made, and the parts cauterised with pure carbolic acid.

If seen after marked symptoms have developed, support the patient with stimulants and quinine. Locally, 5 per cent carbolic acid may be injected into the brawny induration around the seat of inoculation.

No treatment appears to influence the fatal course of the general forms of the disease, but quinine in large doses may be tried. Lately anti-anthrax sera have been tried on the Continent. The serum of Sclavo has given encouraging results. Prophylactic treatment consists in preventing anthrax in animals and preventing the spread of infected material. The bodies of infected animals should be buried unopened.

HYDROPHOBIA—RABIES.

An acute specific disease due to the inoculation of a specific poison generated in animals suffering from rabies.

Ætiology.—The specific organism of rabies has not yet

been discovered. The disease is almost invariably contracted from the bite of a rabid animal, usually the dog; but the cat, wolf, and fox may also transmit it. The virus is in the saliva, which may be infective for a day or two before symptoms have developed. By no means all who are bitten are affected; wounds on uncovered parts are far more dangerous than wounds through clothing. Bites on the face lead to specially acute symptoms.

The average incubation period is from six weeks to two months. It may be as short as a fortnight, or as long as three months.

Morbid Anatomy.—The main feature of rabies is hyperæmia and congestion of the central nervous system. There is also congestion of the pharynx, œsophagus, and stomach.

Symptoms.—The wound by which the poison was introduced, as a rule, rapidly heals, and for a time nothing happens to attract the patient's attention to the scar. In about six to eight weeks or so, the scar may become painful and nervous disturbances manifest themselves. The patient becomes sleepless, peevish, irritable, and experiences a choking sensation about the throat. When the disease is fully developed there are intense muscular spasms, the respiratory muscles and those of deglutition being specially involved; but a more or less tetanoid condition may be observed of nearly all the muscles. There may be opisthotonos. The features may be horribly contorted or wear an aspect of extreme terror; the saliva is not swallowed, and the collection in the mouth of this, and of thick mucus from the congested fauces, causes noisy attempts at ejection, attended with great difficulty.

The face is usually flushed or livid during the attacks, and there may be raving delirium, delusions, and hallucinations. It should be noted that, though the patient is very thirsty, he is afraid to drink, as any attempt at swallowing brings on the spasms at once; even the sound of running water will excite the attacks. There is generally fever, the temperature ranging from 100° to 103°. After from two to three days the patient

may pass into the "paralytic stage," which, however, is more common in animals. He generally dies of exhaustion in from two to ten days after the development of the characteristic symptoms.

Treatment.—The wound when inflicted should be at once sucked, if the mucous membrane of the mouth be unbroken; ligature when possible *above* the bite, cauterise the wound or excise the injured part entirely. The Pasteur treatment should be commenced as soon as possible. It is practically certain to prevent the disease if begun within a week of the bite.

Where there is doubt that the dog was rabid, *it should not be killed*, but isolated and kept under observation for a few days.

When the disease is developed, treatment is merely palliative. Morphia and chloroform for the spasm, and cocaine to diminish the sensitiveness of the throat, are the best remedies.

Pasteur's Method.—A strong virus is obtained by successive cultivations of specially inoculated rabbits. When a virus is procured which will cause madness in *the shortest possible period*, certain portions of the prepared or inoculated spinal cord are exposed to the air. The air has the power of destroying the virus if it be long enough exposed, consequently the virulence or strength can be graduated by the length of time exposed. Pasteur found that by inoculating with an extremely weak virus at first, he could, by successive inoculations of stronger but graduated virus, render the patient incapable of being affected with this disease. For this purpose emulsions of the spinal cord are injected, beginning with one which has been exposed to air for fourteen days, and continuing till a virus of only three days' exposure is reached. In other words—*the necessary soil* for the development of rabies is used up by injections of graduated strengths of the virus. As this can be done during the ordinary incubation period after a bite from a rabid animal, *by the time the*

symptoms of rabies are due, as it were, the soil is no longer suitable for the poison to develop; hence no bad results follow the wound first inflicted. The treatment occupies a fortnight. If begun less than a fortnight before symptoms may be expected to manifest themselves, it is likely to be ineffective. It bears the same relation to hydrophobia that vaccination does to smallpox in those who have been exposed to infection.

TETANUS or LOCK-JAW.

A specific infective disease dependent on the presence and multiplication of a special bacillus, and characterised clinically by severe tonic spasms of the muscles, especially those of the jaw.

Ætiology.—It most often occurs after trivial injuries to the hands or feet. It has frequently occurred in epidemic form amongst newly-born children (*tetanus* or *trismus neonatorum*). When idiopathic in its origin, there is generally a history of sleeping on damp and infected soil. The organism is known as the “bacillus of Nicolaïer,” who has cultivated it from certain putrefying fluids and surface soil. The bacillus is a slender rod, which may grow into long threads, is mobile and anaërobic. A rounded spore is often situated at one end (“drum-stick bacillus”). The organism multiplies only in the wound, the blood and tissues remaining free. The process is therefore primarily local, the systemic effects being due to the absorption of the poison manufactured in the wound. This is a tox-albumin of extraordinary virulence. It appears to travel to the central nervous system along the axons of the motor nerves.

Morbid Anatomy.—Hyperæmia of the cerebral nervous system and small hæmorrhages into the motor centres are the only important changes.

Symptoms.—Usually within ten days of the injury, the patient complains of stiffness in the neck and muscles of the jaw. Gradually lock-jaw (*trismus*) is produced by the tetanic

spasm of the muscles; the eyebrows at the same time become elevated, and the angles of the mouth drawn out, causing the so-called *risus sardonicus*. Soon all the muscles may be affected with paroxysms of tetanic spasms. The body may be arched, the patient resting on his head and heels (opisthotonos); pleurosthotonos or emprosthotonos may sometimes occur. The pain amounts to agony during the paroxysms. Complete relaxation does not occur even in the intervals. The patient may be bathed in sweat, and the temperature reaches sometimes as high as 110° or 112° before death. Acute cases usually die within four days; the more chronic and milder cases may recover. The longer the incubation period, the greater is the chance of recovery.

Diagnosis.—A diagnosis must be made from poisoning by strychnine. It is difficult to confound tetanus with strychnine poisoning, for the latter never causes the jaws to *remain* tetanically closed between the spasms, nor are the muscles of the jaws involved early.

Treatment.—Darkened room—absence of any source of irritation. During the severe paroxysms, give chloroform; between, administer hypodermic injections of morphia, hyoscine, curara, or atropine. The patient may be fed, per rectum, with pancreatised meat extract, etc. The tetanus antitoxin has not proved of much use in acute cases. It should be given hypodermically in repeated doses of 100 cc. It is said to be more effective if given by the intravenous method.

If tetanus is feared, repeated hypodermic doses of 20 cc. may be given. It has been successful in the chronic form, but many of these cases tend to recover spontaneously. The wound should be excised, cauterised, and treated antiseptically.

GONORRHOËAL INFECTION.

In the course of a case of gonorrhœa, as a rule in the acute stage, but sometimes even when it has become chronic, general

symptoms may develop which indicate the spreading of the infective process (*a*) by continuity of tissue, (*b*) by general systemic infection. In class (*a*) are included various forms of disease of the genito-urinary organs, such as salpingitis, endometritis, cystitis; and sometimes by extension along the ureters, pyelitis, or pyelo-nephritis, which, when due to gonorrhœa, is of very grave significance.

Into class (*b*) there fall forms of disease very similar to those which are found in septic conditions (see *Septic Diseases*, p. 52). Occasionally there may be severe and rapidly fatal general infection, as in a case recorded by Osler, which ended fatally fourteen days after the onset of gonorrhœa. Such cases are associated with foci of suppuration in the urinary tract. More frequently local lesions are to be found—

1. In the heart; ulcerative endocarditis or pericarditis :
but oftenest—
2. *In the joints*; gonorrhœal arthritis.

In not a few of these cases the gonococcus has been found in the blood.

Gonorrhœal Arthritis.—This affection is much more frequent in the male than the female. The amount of effusion into the joints is not great, but there is considerable peri-articular œdema. The effusion is seldom purulent, but is more like that of synovitis. There is often much tenderness. Recovery is slow, and there is a liability to the formation of fibrous adhesions, which may lead to ankylosis. Usually several joints are attacked, but sometimes only one. Those which commonly escape in acute rheumatism (sterno-clavicular, intervertebral, etc.) often suffer in “gonorrhœal rheumatism.” The gonococcus may be present in the joints affected. The following forms may be distinguished :—

1. *Polyarthritic*, resembling subacute rheumatism.
2. *Acute arthritis*; sudden involvement of one joint, with severe pain and great swelling.
3. *Chronic hydrarthrosis*; one joint only attacked, oftenest the knee; no pain or redness.

4. *Bursal and synovial form* ; bursæ and tendon-sheaths are mainly affected.
5. *Septicæmic* : intense symptoms of sepsis along with the arthritis.

Diagnosis.—The numerous other causes of synovitis must be excluded. The history of the case, the presence of a urethral discharge, and the obstinacy of the symptoms, will be of service.

Treatment.—There is no specific remedy. Iodide of potassium is much vaunted, but is often useless. General tonic treatment must be combined with local measures. The urethral discharge must be treated; the joints, if acutely affected, must be kept at rest, while in chronic cases massage is useful. In some cases the thermocautery, in others, local baths of superheated air may do good. In obstinate cases incision and irrigation of the joints is the best treatment.

SYPHILIS.

A specific infective disease of slow evolution and long duration, resulting from (1) impure venereal intercourse; (2) inoculation with the virus obtained from syphilitic lesions; or (3) transmission through diseased parent or parents—*i.e.*, congenital syphilis. Syphilis is such an extensive subject, and so highly important a factor in many diseases, that the reader is referred to a monograph on the subject.

Modes of Infection.—Infection may be direct (sexual congress, finger of the accoucheur, tattooing, etc.) or mediate, through contaminated instruments or drinking-vessels. The secretions of the chancre and of secondary lesions, and the blood in the secondary stage, all contain the virus. Tertiary lesions are probably not infective.

The disease may also be transmitted hereditarily, either through the father or the mother. Though the mother of a syphilitic child may show no signs of the disease, she has acquired immunity from it and cannot be infected (Colles' law). The lesions of congenital syphilis are infective.

Bacteriology.—Until quite recently no specific organism was known, although various organisms had been described, the most familiar being the bacillus of Lustgarten. But in 1905 Schaudinn and Hoffmann discovered the *spirochaeta pallida*, and somewhat later Siegel described another protozoon, the *cytorrhcytes luis*, which may possibly be a developmental stage of the former. The spirochaeta is constantly present in the primary and secondary lesions, and in hereditary syphilis. It is scarce in tertiary lesions, although in a few cases it has been found in gummata. In the secondary stages it is also present in the blood and the viscera. It is not found in non-specific lesions. Although it has not been cultivated, it is now very generally accepted as the specific cause of syphilis.

Characters.—The spirochaeta pallida (*treponema* or *spironema pallidum*) is a very delicate protozoon, mobile and spiral, the turns of the spiral being arranged corkscrew-fashion. It is from 4 to 14 feet long, and only $\frac{1}{4}$ foot thick. It is only slightly refractile, difficult to stain, and in each spiral there are from eight to twenty-four turns. It possesses a flagellum at each end, and is devoid of undulatory membrane. In these respects, and in its smaller size, it differs from other spirochaetae, notably the spirochaeta refringens, which is found on the surface of lesions of the genital organs.

Pathology of Syphilis.—The primary chancre is largely an accumulation of round cells in the corium with increase of connective tissue. The endothelium of the blood vessels is swollen, and proliferates, their walls are infiltrated with round cells, and their lumen is narrowed. The adjacent lymphatic glands are also infiltrated with round cells.

In the secondary stage there are cutaneous lesions with changes of a similar type. The vessels are also implicated. There may be macules, papules, pustules, etc. "Mucous patches" are papules modified by their situation on moist skin or mucous membrane. Where growth is exuberant, especially around the anus, and on the scrotum or vulva, they are called condylomata.

The tertiary stage is characterised by the presence of *gummata*, hard, nodular, localised masses *adherent to the surrounding tissues*. They may be quite small, or even larger than a hen's egg. They consist of round cells with epithelioid cells and occasional giant cells. They are surrounded by firm connective tissue. They tend to necrosis in the centre, and may discharge through skin or mucous membrane. When they affect the viscera they may cause much cicatricial deformity.

Profound arterial changes may occur in the later stages of syphilis, favouring the development of aneurysm, atheroma, cerebral thrombosis, etc.

Briefly put, the following are the more important facts of acquired syphilis—four periods are described—(1) Incubation ; (2) Primary Sore ; (3) Secondary Symptoms ; and (4) Tertiary Stage.

1. *Incubation Period* ; varies from two to six weeks, usually between three and five.

2. *Primary Sore*, or chancre, is a papule with an *indurated* base. The surface may or may not ulcerate, but is usually depressed in the centre. The hardness or induration extends to the nearest lymphatic glands, and ultimately all the lymphatic glands may become involved. The glands are freely movable under the skin ("rolling glands"), discrete, and not painful. They rarely suppurate. The chancre or "*hard sore*" finally disappears, leaving a cicatrix behind.

3. *Secondary Symptoms*.—After the appearance of the primary sore there is an interval ("second incubation") varying from one to three months, but usually lasting about six weeks, before secondary symptoms appear. These are as follows :—

FIRST STAGE.

- (1) Syphilitic fever, with transient hyperæmia of the skin, roseola, etc.
- (2) Sore throat.

SECOND STAGE.

The second stage is marked by skin eruptions (syphilides), clinically distinguished by—

- (1) Their symmetry.
- (2) Copper colour, dull red at first, afterwards a reddish brown.
- (3) A tendency to circular form of patches.
- (4) The scales when present are light and small.
- (5) Ulceration is common—sharp edges and ashy-grey surfaces.
- (6) The crusts are greenish and thick, adhering firmly, and often limpet-shaped (rupioid).
- (7) The eruptions are often mixed, or polymorphous.

The other changes are—

“Mucous patches” in the mouth, and condylomata—soft, flat, warty growths with a greyish secretion around the anus, on the scrotum, or in the groins.

Ulceration of throat.

Syphilitic iritis.

Syphilitic periostitis.

Anæmia.

Loss of hair (alopecia) *not* followed usually by permanent baldness. Nocturnal headache.

4. *Third or Tertiary Stage.*—The anæmia has become more marked, and the manifestations of the disease are those of serious malnutrition. Cutaneous syphilides are not symmetrical as in the secondary stage, but scattered, and they tend to form deep ulcers, which leave well-marked scars. The bones are often affected, and periosteal “nodes” are common on the long bones and the calvarium. Syphilitic endarteritis is common, and may be the cause of aneurysm or of cerebral thrombosis. Deep-seated organic changes due to the formation of gummata are also very common. These growths may be found subcutaneously in the muscles, or in any of the internal

organs. They are met with in the brain (Jacksonian epilepsy), bones, testicle, and liver (syphilitic cirrhosis), etc., etc. Ultimately they may be gradually absorbed, thus causing cicatricial deformity of internal organs; or if they are superficial, they may ulcerate and discharge upon the surface. According to some, almost any chronic disease may be caused directly or indirectly by syphilis. Whilst no doubt syphilis is often a most powerful or even the chief factor in many diseases, and its importance cannot be over-estimated, still, probably, it is often unjustly held responsible for many serious lesions. There are, however, certain chronic diseases which have a very definite connection with syphilis. These are often referred to as para-syphilitic diseases, *i.e.* besides the direct action of the microbe on the blood-vessels and connective tissue, it acts by its toxins on the cells themselves, giving rise to degenerative changes. Chief among them are amyloid disease, locomotor ataxia, and general paralysis, epilepsy, and in hereditary syphilis, infantilism.

In women, miscarriages or still-births are frequent. Should the child survive, it may be the subject of congenital syphilis.

The secondary stage lasts from six months to a year; the tertiary follows it from a year to three or four years after infection. Tertiary lesions are obstinate, and tend to relapse after treatment.

Tertiary syphilis is neither inoculable, with the possible exception of the secretions from discharging gummata, nor transmissible to offspring.

Congenital syphilis may originate—(1) from father; (2) from mother; (3) from both. If the father alone be syphilitic, then the sequence may be—

1. Father.
2. Child.
3. Mother through child. In these cases the mother is immunised, but may present no signs of the disease (Colles' law).

Symptoms.—

1. The child may be born syphilitised, or may be apparently healthy, and develop “snuffles” within the first three months.
2. Develops a peculiar “old man” appearance.
3. Depression at root of nose from necrosis of nasal bones.
4. Mucous tubercles at the mouth or anus.
5. Red patches on buttocks, ankles, or hands.
6. Subacute onychia, fissures at lips, and later, notched “Hutchinson” teeth, keratitis or iritis, bone lesions, and gummata.

Treatment.—The two specific drugs are mercury and iodide of potassium, the first being of the greatest value in the earlier stages, the second in the later.

The treatment of the primary sore consists mainly in local cleanliness. If the diagnosis is *absolutely certain* (it is made certain by the discovery of the spirochaeta, although a negative result does not exclude syphilis), mercury may be commenced at once, and may avert even secondary symptoms; but it is often advisable to wait till these appear before commencing specific treatment. This consists in the early stages in the administration of one or other of the preparations of mercury. The preparations administered, and the methods of administration, are too numerous to describe in full. In the general run of cases Hutchinson's pill (Hydrarg. c. Cretâ gr. i, Pulv. Ipecac. Co. gr. i. Sig. one or two thrice daily) is very effective. It can be given for long periods without producing salivation or diarrhœa. Treatment must not be stopped when symptoms have disappeared, but *must be continued for eighteen months or two years*. Where symptoms are more urgent, and a rapid effect is desired, inunction of blue ointment, \mathfrak{z} i daily, or hypodermic injection of the perchloride, gr. $\frac{1}{8}$ every second day, may be practised. The condition of the mouth must be looked to, and smoking should be forbidden if there are lesions in the mouth. In the tertiary stages, iodide of potassium to a great

extent takes the place of mercury, but the two are often advantageously combined. Doses of 5 to 20 grains thrice daily usually suffice, but in cerebral syphilis the drug must be rapidly pushed to even larger doses.

In cases of pregnancy the mother must be actively treated if the father be syphilitic, even though she herself is apparently healthy. Infants suffering from congenital syphilis are best treated with grey powder (gr. $\frac{1}{8}$ - $\frac{1}{3}$ thrice daily), or in severer cases by inunction.

In every case, besides treating the specific disease, the general health must be sedulously cared for. Anæmia may be treated by iron or arsenic, the general strength built up by hypophosphites, cod-liver oil, etc., and well-to-do patients are often rapidly improved by a course at such a spa as Aix-les-Bains.

TUBERCULOSIS.

A specific infective disease, produced by the action of the *bacillus tuberculosis*, and characterised by the formation of "tubercles," which tend to undergo degenerative changes in the direction of softening or of calcification. The disease may be local or generalised (acute miliary tuberculosis); in the local forms the lungs are oftenest involved, but any organ may be attacked.

Ætiology.—Those predisposed to tuberculosis are frequently of feeble physical development, though even the athletic are often attacked. A special type of thorax—the alar chest—often accompanies the pulmonary form (see Phthisis). The sexes are almost equally liable. No age is exempt, but it is relatively less common in the old. Phthisis is a disease chiefly of adolescence and adult life; children oftener suffer from tuberculosis of bone, glands, or meninges.

Tuberculosis is not hereditary in the direct sense, although in rare instances infants have been born tuberculous, and the bacillus has been found in the placenta. But a *predisposition* to the disease is probably inherited; the tissues of the offspring

are less resistant to the attack of the bacillus. Those of tuberculous family history are less likely to respond favourably to treatment, if attacked, than those in whom the disease is sporadic.

Modes of Infection.—1. By inoculation of the tuberculous material (lupus, post-mortem wounds). The process usually remains local.

2. *By inhalation of the dried and powdered sputum* (dust of apartments, etc.). Crowded and ill-ventilated dwellings, and sedentary occupations thus predispose to the disease.

3. By ingestion of tuberculous material, (*a*) by swallowing the sputum; or (*b*) by swallowing infected meat or *milk*. Koch has lately denied that animal tuberculosis can be transmitted to man, but the result of the recent Commission appointed to investigate the subject has been fully to establish its infectivity for man.

The bacillus is a small, motionless, rod-shaped organism, 3 to 4 μ in length, and often slightly curved. It is not mobile, and has no flagella. It stains slowly with the aid of heat, and is difficult to decolourise by acids. The appearance of beading often noticed is probably due to the presence of vacuoles, and not to spore-formation. The organism grows best on sterilised blood-serum at 37° C. Growth is arrested at 28° C. on one hand, and 42° C. on the other.

Morbid Anatomy (general).—The tubercle from which the disease gets its name is a small body visible to the naked eye or with a lens. At first it is semi-transparent, grey, and gelatinous in appearance, later it becomes opaque, softened, and yellowish in the centre. Tubercles may be few or very numerous in the affected organs, and may be isolated or clustered in masses.

Microscopically they show from within outwards—

1. A central giant cell, believed by some to be due to the fusion of several epithelioid cells.

2. A layer of epithelioid cells.

3. A zone of lymphocytes, and often a dense fibrous capsule.

There are no blood-vessels in the tubercle, and therefore it is open to degenerative changes. The cells become fatty and soft, the softened material being discharged in sputum, urine, or stools, or there may be a conservative reaction, leading to the encapsulation (calcification) of the *materies morbi*, and to the survival of those affected with obsolete tubercle. Too often the advantage is on the side of the attack.

The bacilli are found in varying numbers in the giant cells and the epithelioid cells.

There is nothing distinctive in the early processes of tubercle except the discovery of the bacillus. Later, secondary inflammatory processes occur around the tubercles. There are several methods by which tubercle may spread within the body of an infected patient:—(1) along mucous membranes (pharynx to Eustachian tube, middle ear, and meninges, etc.); (2) by swallowing his own sputum (intestinal ulcers); (3) by the lymphatics; and (4) by the blood. All these demand a local infection for a starting-point. But general tuberculosis, with which alone we deal just now, is usually started by a local tuberculous process, invading the blood-vessels, and spreading all over the body.

ACUTE MILIARY TUBERCULOSIS.

Ætiology.—Much more frequent in early childhood; not uncommon between twenty and thirty. Usually secondary to chronic tuberculous processes in internal lymphatic glands or bones. It may follow other infective diseases—measles, whooping-cough, enteric.

There are three main types,—acute generalised miliary tuberculosis, acute pulmonary tuberculosis, acute meningeal tuberculosis. The last will be described under Diseases of the Nervous System.

1. *In acute generalised miliary tuberculosis* tubercles are thickly scattered through all the organs. They are small, miliary or sub-miliary, and very numerous.

The symptoms are those of a profound general infection, and the disease is often mistaken for enteric fever (hence called *typhoid* form). It begins with increasing weakness and anorexia, and after some time irregular fever appears. It may be continuous, remittent, or intermittent; oftenest continuous, with a difference of about 2° between morning and evening temperatures. The pulse is rapid and feeble, the tongue dry, the cheeks flushed. Delirium is marked, and usually of the typhoid type, deepening into coma. The pulmonary symptoms are those of slight bronchitis, or less commonly broncho-pneumonia. The respirations are rapid at first. There is often cyanosis. Cheyne-Stokes breathing may appear later.

The diagnosis from enteric fever is often difficult. The most important points in favour of tuberculosis are—

- (1) Irregularity of the fever.
- (2) Comparative rarity of diarrhœa.
- (3) Absence of rose-spots.
- (4) Absence of enteric bacillus in blood.
- (5) Widal's reaction is negative.
- (6) Possible presence of tubercle in the choroid.

II. *The pulmonary form*.—The nodules are most numerous in the lungs, occurring either in groups or universally diffused. They are thickly set beneath the pleura. The lungs are congested, and often emphysematous. Collapsed patches, and areas of consolidation of varying size are to be found.

Symptoms.—The onset is usually sudden, with perhaps a previous history of cough or chronic phthisis. In children measles or whooping-cough may have gone before. Breathing is rapid, and dyspnœa marked. Cough may not be very troublesome. The spit is mucous in character, but may be muco-purulent or rusty. Cyanosis is prominent. Fever is well marked (102° - 104°). The physical signs are those of diffuse bronchitis and acute emphysema—hyper-resonant percussion, varying intensity of breath-sounds, presence of sibilus or rhonchus, and medium crepitations. There is sometimes diminished resonance at the bases (broncho-pneumonia).

The duration of the disease varies from one to several weeks. It is generally fatal.

The diagnosis is assisted by the history, suddenness of onset, and possible discovery of the bacillus in the spit. When the bacillus is present, however, it is due to the presence of a primary focus of chronic tuberculosis in the lung. In the acute process the newly formed tubercles have rarely time to break down and discharge their organisms in the sputum before the patient's death.

Treatment in both forms is purely symptomatic. Recovery is exceptional.

Local tuberculosis will be dealt with under Lungs, Alimentary Tract, Nervous System, etc.

RHEUMATIC FEVER (ACUTE RHEUMATISM).

An acute febrile disease characterised by poly-arthritis, a tendency to hyper-pyrexia, a special tendency to involve the pericardium and endocardium, and marked anæmia.

Ætiology.—The larger number of cases can be traced to exposure to cold, inclement weather, etc. It is most common during spring, in humid climates, and affects *young* adults most frequently. Males are more often attacked than females, although before the age of twenty it is more common in women. The influence of heredity is often definitely noticeable.

Pathology.—The true cause or causes of rheumatic fever are still unknown. Though there is little doubt that the disease is of microbic origin, so far, out of the numerous organisms (chiefly cocci) which have been described, none has been absolutely proved specific. It has been held that the disease is due to excess of lactic acid in the blood, or to nervous influence, but these theories are now practically discredited. The disease shows a distinct tendency to epidemic prevalence at irregular intervals. Its clinical course is strikingly similar to that of

septic infections. No immunity, but rather an increased liability, is conferred by previous attacks.

The organism which is regarded as the most probable cause of the disease is the *diplococcus rheumaticus* of Poynton and Paine, which is found in the blood and synovial fluid, and also in the valves of the heart, when these are affected. The poison causes a rapid destruction of red corpuscles, resulting in anæmia. There is a moderate leucocytosis.

Morbid Anatomy.—The synovial membrane undergoes the changes common to inflammation of serous membranes—viz., hyperæmia, exudation of lymph, and effusion of fluid. The fluid is usually turbid, but seldom becomes purulent. The ligamentous structures and synovial membrane are swollen, and often the cartilages are slightly eroded. The blood, though it contains an excess of fibrin, clots more slowly than normal; this, however, occurs in many other conditions.

The fluid of the secondary affections such as pericarditis or pleurisy, and the vegetations of the endocarditis, contain the diplococcus and, it may be, pus organisms. There is nothing to differentiate these from other inflammations. The myocardium is very commonly affected.

Symptoms.—After a feeling of malaise, more or less soreness, and general stiffness, and very often after an attack of tonsillitis, the rheumatic pain begins in one of the larger joints: usually the knee, wrists, or ankles.

The arthritis extends very rapidly to any of the medium-sized joints. At first the affected joints are red, hot, swollen, and intensely painful; later, the redness becomes less marked, and the joint may assume a dead-white appearance. Frequently the inflammation rapidly subsides in one joint only to appear in another.

The pulse is rapid, full, and soft.

The temperature may be very high, even to 107° ; indeed, hyperpyrexia is not uncommon in this fever. In the bulk of cases, however, it remains between 101° and 103° . It usually reaches its height in twenty-four hours, and is very irregular.

Marked sweating of a peculiar sourish smell is a constant feature, and the various hair follicles and other cutaneous glands may become inflamed and painful in consequence.

As the disease advances *anæmia becomes very pronounced*. This anæmia may be increased by the salicylates, but is mainly due to the rheumatic poison. There is well-marked leucocytosis.

The tongue is usually very large, flat, and covered with such an extremely thick fur that it has been named the "blanket" tongue.

The urine is scanty, highly coloured, and often loaded with urates. The chlorides are diminished or absent.

Murmurs, either hæmic or organic, may often be heard over the heart, which should be examined every day.

The more common complications are—

1. Endocarditis, most frequent in youth, and affecting oftenest the mitral valve. It occurs in about half the cases.
2. Pericarditis, much less frequent.
3. Myocarditis, which may be slight or profound.
4. Pleurisy.
5. Hyperpyrexia, often attended with delirium or coma.
6. Certain skin eruptions, such as sudamina, miliaria, "pelioses" or small red petechial spots around the ankles, and purpura.
7. Pharyngitis and tonsillitis.
8. Meningitis (very rare).

Frequently subcutaneous fibrous nodules develop over bony ridges. Though rarely fatal, and though the severe symptoms usually subside in about fourteen days, no disease is more often attended with relapses and grave results. Valvular diseases of the heart, etc., constantly follow in its wake, and sometimes chorea.

Diagnosis.—The distinction between acute rheumatism and septic polyarthritis is sometimes difficult. In the latter there is evidence of a primary septic disease (gonorrhœa, puerperal

fever, etc.), and a much greater tendency to pus formation, and to marked oscillations of temperature.

Treatment as regards the general management. See that there is absolute rest, perfect protection of the limbs by wrapping them up in cotton-wool, and support the bed-clothes by cradles. The diet should consist principally of milk and soda-water; beef-tea and chicken may be allowed later on. The patient should wear a flannel night-dress, and sleep between blankets.

As regards treatment, **undoubtedly the salicylates rank first nowadays**. There can be no two opinions as to their efficacy in easing pain, but from the experience of fourteen well-marked and well-watched cases, I share the belief of that school which attaches more value to the old alkaline treatment—*i.e.*, letting the patient drink *ad libitum* of citrate or bicarbonate of potash, with an occasional dose of quinine, and the administration of pulv. ipecac. co. to ease the pain (A. W.). While this was Dr. Wheeler's view, and is one still held by many, the majority consider that the salicylates shorten the course, protect the heart, and lessen liability to relapse. Salicylate of soda or salicin, gr. x-xx for an adult, should be given every two to four hours till pain is relieved, and afterwards every four hours till the temperature is normal. Should vomiting or ringing in the ears occur, the dose must be reduced. After the normal temperature is reached, the dose should be gradually lessened. Salicin is the better drug for children, as being less depressant. There is no reason why alkalies should not be given along with it, or with the salicylates (W. R. J.). A solution of the salicylates locally as a lotion or fomentation is of the utmost value. Pot. iodid. may be combined with salicylates in obstinate cases. Treat complications on rational principles already laid down. For hyperpyrexia more active measures are required, such as the cold bath, cold packing, or sponging with ice-cold water. During the process the temperature should not be allowed to fall below 101°, as the fall continues for some time after the patient is restored to bed.

CONSTITUTIONAL DISEASES.

GOUT

Is a disorder possibly nutritional in its character, associated with an excessive amount of uric acid in the blood, and deposits of biurate of sodium in the joints. Clinically, it is manifested by periodical attacks of acute arthritis, certain visceral disturbances, and later, deformity of the joints attacked.

Ætiology.—1. Heredity is certainly a most important factor, and the disease is curiously developed in the grandchildren. It is more frequent in the male, and it occurs chiefly in middle or later life, although it is not unknown about the age of puberty.

2. Alcoholism, especially indulgence in sweet wines, and heavy malt liquors. Strange to say, Italians who drink large quantities of light wines, and Scotsmen who certainly freely indulge in good whisky, are singularly free from gout.

3. Gluttony. This is as important a factor as excessive drinking, especially when combined with a sedentary life.

4. Certain poisons ; such as lead, etc., which tend to decrease the metabolic activity of the body.

5. Insufficient food (the poor man's gout). This condition, however, is associated with defective hygiene, and only too frequently, with excessive consumption of poor or raw spirits, etc.

Pathology. In no disease can we point to a more constant

feature than excess of uric acid in cases of gout. This salt is found in excess in the gouty blood, in the gouty joints, and in the exuded serum of the gouty arthritis ; therefore there cannot be the least possible doubt that the disease is due either to the accumulation of uric acid in excess, or to *those conditions which give rise to the excessive formation of uric acid*. Now, if we were to inject uric acid into the blood of a fairly healthy individual we would not produce gout ; and, moreover, von Jaksch and others show that uric acid may be always produced by deficient oxidation, and that excess of uric acid is common in many diseases, such as leucocythæmia, pernicious anæmia, etc. ; yet gouty arthritis is by no means a common complication in these diseases, therefore there must be a “something more” than mere uric acid in excess to cause such a condition as gout.

Sir Dyce Duckworth assumes, “There is a basic arthritic stock—a diathetic habit of which gout and rheumatism are two distinct branches. The gouty diathesis is expressed in (1) a neurosis of the nerve centres, which may be inherited or acquired ; and (2) a peculiar incapacity for normal elaboration within the whole body, not merely in the liver or in one or two organs, of food, whereby uric acid is formed at times in excess, or is incapable of being duly transformed into more soluble and less noxious products.” (Sir Dyce Duckworth’s *Handbook on Gout*.) According to such an eminent authority, gout means something more than uric acid in excess.

Sir William Roberts has shown that *normally* uric acid exists as a quadriurate which requires an alkaline medium in order to be kept in a perfectly soluble condition. In other words, the quadriurate is deposited in a *weak* alkaline medium as a biurate compound, a salt which is found in the joints affected by gout. I would suggest the following statement as representing our present knowledge of the pathology of gout :—Mere excessive formation of uric acid within the body is either the outcome of excessive nitrogenous (or proteid) consumption, or of deficient oxidation on the part of the tissues (liver, etc.) This condition constitutes the “lithæmia” of the older writers, and is one

highly predisposing to gout. Now, if we assume that through certain changes, nervous or otherwise, the blood, or certain tissues, become less alkaline than is sufficient to hold the quadriurate in solution, a precipitation of a more insoluble salt, viz., the biurate, must occur; and, if such a precipitation takes place in the joints, no wonder that acute arthritis is excited thereby. Excess of the quadriurate may be physiological and compatible for a time with health; *deposit of the biurate on the other hand, is pathological, and results in gout.* This seems rational when we remember that the circulation in the smaller joints is very sluggish, and consequently such parts are prone to low inflammatory processes, resulting in a local diminished alkalinity of the blood, thus favouring the local precipitation of the circulating quadriurate. The consequent arthritis and febrile disturbance set up further explain why gouty patients feel so much better after an attack of gout, for the increase of metabolism of the febrile state, and the alkaline treatment, all tend to burn up as it were, or promote the excretion of the excessive uric acid in the blood. The restricted diet also gives the liver a holiday, and enables it to perform its functions with renewed vigour.

Further, our theory harmonises with the suggestion that gout is more or less of a neurotic origin, for we pre-suppose *some* devitalisation of the tissues and joints, and joints are notoriously influenced by atrophic nervous changes, as witness Charcot's joint, etc.

Bain's view is that though the uric acid phenomena form an important incident in the pathology, the essential factor has not yet been discovered. He considers that the disease originates in the alimentary tract, "from excessive formation there of a purely physiological substance, or more probably from the production of an abnormal substance" by deranged digestive processes or by microbic action.

Let us remember one thing more, that once a joint is affected with gout, if the deposit is not all cleared away, like a calculus in the bladder, it simply forms a nidus for the formation of a large concretion.

Morbid Anatomy.—The metatarso-phalangeal joint of the great toe is most often affected. If we examine the joint at various stages, we find the following conditions—

A deposit of fine crystalline needles apparently in the *superficial* parts of the cartilages, but really interstitial; next, the synovial membranes, cartilages, and ligaments become covered with a chalk-like deposit of biurate of soda. Underneath the deposit, the tissues are in a more or less necrosed condition. The synovial fluid contains crystals of uric acid.

Later, the cartilages may be eroded, and the synovial membranes thickened. The ends of the bones are enlarged and the joints deformed. Nodular masses around the joints, consisting of collections of biurate of soda *plus* calcium phosphate, form the so-called chalk stones or *tophi* of chronic gout, which may ulcerate through the skin. These deposits may occur elsewhere than in the joints, as in the lobes of the ear, tendinous aponeurosis of muscles, sclerotic of the eye, etc. Garrod believes these concretions are merely calcification of gouty inflammatory products, and no doubt he is right. Interstitial nephritis (cirrhosis of the kidney) is common in gout, and is associated with deposits of the biurate in the intertubular tissue. Arteriosclerosis, leading to hypertrophy of the left ventricle, is also common.

Symptoms of Acute Gout.—Usually there are some premonitory symptoms, such as giddiness, mental depression, flatulence, irritability of temper, scanty and high-coloured urine, etc. The attack most commonly commences in the early hours of the morning, with severe pain in the big toe. The pain increases to acute agony in some cases, rendering sleep or rest out of the question. The patient becomes very feverish, or he may feel chilly, and shiver violently without any rise of temperature. The joint at first is bright red, and exquisitely tender; later, it is more swollen, and of a livid or dull dusky red, with distended venules standing out. The swelling extends some distance from the joints. The skin desquamates in thick flakes when the attack is over. An attack lasts from

five to twelve days, but the severe pain is not constant, or rather there are lulls, with exacerbations of severe pain at intervals of the day, especially at night-time. The urine at first is scanty and high-coloured *but the uric acid is diminished in amount*; later, the excretion of *uric acid is very much increased*. During the attack the patient usually is most irritable, the tongue is furred, the breath may be offensive, he has no appetite, and the bowels are constipated. Subsequent attacks may affect the joint first implicated, or a number of joints may become involved. If the attacks are fairly frequent, they cause the so-called chronic gout; that is, a condition recognised by a characteristic deformity of the joints, important changes in various organs, especially the red granular cirrhotic, or "gouty kidney."

No disease is quoted more frequently than chronic gout as an important aetiological factor in the causation of other diseases. Such a statement, however, is largely the outcome of habit and fashion. Many eminent men employ the terms, "gouty habit," "specific taint," in a ridiculously loose fashion. It is sometimes actually refreshing to hear a nervous disease described as *not* dependent on one of these causes.

Chronic Gout.—Must not be confounded with rheumatoid arthritis, though the sufferer may be crippled in much the same manner.

The small joints of the toes and fingers are most often affected. The fingers are frequently deflected to the ulnar side. These joints are often distended by "tophi," which at first lie under the skin, but may ulcerate through.

The kidney undergoes a marked cirrhotic change (see Chronic Bright's Disease). In such cases the pyramidal area of the kidney often shows yellowish-white streaks of urate crystals.

Other complications than those of "gouty kidney," are—

1. Oedema of the glottis, which is most easily excited, and is singularly fatal in the gouty state.

2. "Gouty" urethritis, which may resemble a gleet.
3. Renal calculi.
4. Extensive sclerosis of the arteries, and the dangers arising therefrom.
5. Cardiac complications,—palpitation, pain, dyspnœa.
6. Cerebral complications, — apoplexy, congestion of meninges.
7. Retention of urine.
8. Cirrhosis of the liver, with its train of symptoms.
9. Eczema.
10. Chronic bronchitis is very frequent.

Suppressed gout is the condition in which the development of internal symptoms coincides with and may be attributed to inhibition of the joint affection.

Garrod's thread test is designed to detect uric acid in the blood. Take ʒij of blood serum or of the fluid raised by a blister and mix in a watch-glass with ℥xij of glacial acetic acid. Immerse in the fluid one or two ultimate strands from a piece of linen, and leave in a warm place until evaporated to a jelly-like consistency. Transfer bodily to the stage of a microscope, and examine the strands with a low power for uric acid crystals. The test is not very reliable.

Treatment.—During the acute stage—

1. Absolute rest, elevation of the limb, and hot fomentations to the joints affected.

2. Diet to consist of milk and potash water, or at least *non-nitrogenous* food.

3. Medicinal treatment. — Clear out the bowels with five grains of calomel, followed by a full dose of Carlsbad salts. It is important to secure a daily motion, and for this aperient mineral waters are very useful. Give a mixture containing colchicum, iodide of potassium, bicarbonate of potassium, and hyoscyamus. Iodide of potassium, with laudanum and salicylate of soda added to the hot fomentations, speedily relieves the painful joint.

When the acute attack subsides, massage must be employed, for it is absurd to think "medicines" will remove the biurate deposit from the joints.

Treatment of Chronic Gout.—Careful regulation of diet, avoidance of alcoholic excesses, and proper exercise, are the principal points to remember. Arsenic and strychnine as medicines, lithia water as a habitual beverage, frequent Turkish baths (when they agree), and massage to the affected limbs, go far to complete a cure.

RICKETS.

Rickets is a general disorder, which attacks infants and young children who are subjected to improper feeding, want of sunlight, and other unhealthy hygienic conditions. It is attended by characteristic changes in the development of the bones.

Clinically, it is manifested by wasting, stunted growth, characteristic physiognomy and deformities.

Ætiology.—Occurs mostly among the poor of overcrowded cities. It is occasionally seen among the offspring of the wealthy in whom there is a taint of syphilis or tuberculosis. There cannot be any doubt that many cases of congenital syphilis subsequently develop rickets, but the relationship is merely coincidental. The *absence of lime salts* in the food must be looked upon as an important ætiological factor, if such absence is prolonged. Often, however, calcium salts are abundantly supplied in very marked cases of rickets. Deficiency in animal fats and proteids is very constantly associated with the disease (Cheadle). It is therefore most common in children who have been artificially fed, or who have been kept too long at the breast. Deficiency of food, faulty hygiene, and bad ventilation are contributory causes.

Pathology.—We must first briefly consider the more constant morbid anatomical characters.

1. Change in bones. The long bones, the ribs, and the skull are chiefly affected.

- (1) Their specific gravity falls, the water and organic matter are increased, whilst *the earthy matter is decreased*.
- (2) The amount of fat is increased.
- (3) They occasionally do not yield normal gelatine.
- (4) The epiphyseal ends show under the microscope—increased vascularity, irregular calcification, small islets of uncalcified matrix, and large spaces due to absorption by osteoclasts; at the junction of the epiphysis with the shaft, the bluish cartilaginous zone is thickened, and very irregular; the bone formed is more vascular and spongy than ordinary bone.
- (5) In the skull the fontanelles remain open, the bones are thin and soft (*cranio-tabes*), the frontal and parietal bones are hypertrophied, the forehead is broad, and the top of the head is flat.

2. The blood changes are a deficiency of red corpuscles, and of hæmoglobin in the corpuscles present; associated with the presence of much granular detritus (Goodhart).

3. Changes in the lymphoid tissues.—The spleen and lymphatic glands nearly always show extensive fibrous changes. The spleen is usually enlarged, and the liver may also be so. This results in enlargement of the abdomen, increased by a consistent dilatation and ectasis of the stomach and intestines.

As rickets is often associated with a too excessive administration of starches, it has been assumed that lactic acid is thereby formed in abundance, and that this excess of acid is the real cause of rickets, by causing solution of the calcium salts. This theory is not widely held.

From a careful consideration of the morbid anatomy, we must assume at present that rickets is due to either—

1. Non-absorption into the blood of lime salts, through some morbid condition of the alimentary canal; or—

2. An inability on the part of the bone-forming tissues to appropriate the lime which is conveyed to them.

The first theory may be usefully contrasted with one of the explanations of chlorosis—*i.e.*, disordered condition of the alimentary canal, interfering with the assimilation of iron.

Symptoms.—The symptoms of a typical case of rickets are very characteristic.

General configuration.—The head is elongated from back to front, the forehead is square and overhanging, the fontanelles are slow in closing, and the veins in the skin are distended. The flat bones are usually much thickened, and, indeed, are frequently “bossed” by irregular masses of calcified material. It is important, however, to remember that very often the occipital bone, and certain parts of the parietal bones are *thinned* out. Though the skull as a whole is enlarged, the puny face makes the enlargement more noticeable by comparison; measurements show the enlargement to be more apparent than real. The brain development may be seriously handicapped by this peculiar deformity, inasmuch as it allows the hind part to develop, but not the intellectual portion—*i.e.*, the frontal lobes.

The ribs often show characteristic “beading” at the junction of the costal cartilages. The thorax tends to become cottage-loaf in shape. “Pigeon breast” is also common.

The lower limbs are bowed, or sickle-shaped, and show the well-marked epiphyseal enlargements, especially at the lower end of the *tibia*.

The upper limbs also show the most marked changes at the lower ends of the ulna and radius, but the humerus, scapula, etc., are also affected.

The pelvis is often much deformed, and this may later add serious complications to parturition.

The spine is frequently distorted.

It will be seen from the above, that the deformities depend partly on excessive deposits of calcareous material, and to a

certain extent on the yielding of the softened bone to mechanical causes; hence the necessity of rickety subjects avoiding any undue or avoidable strain, such as too early walking, sitting in cramped positions, carrying weights, etc.

The abdomen is usually prominent from distension by gas, and the presence of the enlarged liver and spleen.

The other clinical symptoms are—general and marked tenderness of the body, throwing off the bed-clothes at night, profuse sweating, and constipation alternating with diarrhoea. The stools are usually pasty, or green, like boiled spinach, and are intensely foetid at times. These symptoms, taken with the general emaciation and deformities already described, make a diagnosis very easy. Of the complications there is no end, but we must specially mention convulsions, tetany, strabismus, and laryngismus stridulus. Green-stick fractures are not uncommon.

Treatment. —No cases deserve and require more thoughtful and careful treatment. If the mother be unhealthy, a wet nurse, or the “sterilised milk” plan of artificial feeding must be prescribed. Plenty of fresh air, sunny rooms, and gentle massage must be the routine treatment in all cases. The diet for older children must be varied, and an excess of carbohydrates avoided. Carefully planned gymnastic exercises may help to prevent deformities, or cure those already formed. Of drugs, phosphorus, lacto-phosphates of iron, arsenic, iodide of iron, cod-liver oil, etc., all have their advocates. But careful hygiene is of far more value than medicinal treatment. Dr. Palm, of Wigton, points out “that sunlight in the dwelling not only reveals unsuspected dirt, but is nature’s disinfectant as well as a stimulant and tonic.” There can be no possible doubt as to the all-important influence of sunlight to consumers of large quantities of carbohydrates.

CHRONIC RHEUMATISM.

Chronic rheumatism is sometimes a sequel of rheumatic fever, but is generally a separate constitutional affection.

Ætiology.—Is most common amongst the middle-aged poor. Senility seems to be a predisposing cause, amongst both poor and rich.

Pathology.—Is at present ill understood; the fibrous textures around joints, enveloping nerves, or the aponeurotic sheaths of the muscles, periosteum of bones and tendons all suffer more or less. Though these structures become inflamed, very little alteration results—*i.e.*, the joints are rarely deformed, though the ligaments and synovial membranes may become much thickened, and the movements of the joint limited. Bony ankylosis is not met with.

Symptoms.—Pain and stiffness of the parts involved are the main features. The pain is usually worse on movement, but it should be noted that continued exercise mitigates the pain; for example, a man goes to bed with muscular rheumatism, as long as he keeps quiet the pain is not severe, but the slightest movement causes acute suffering. In the morning, on attempting to rise the pain may be very severe, but after working for a time the pain becomes tolerable though it does not disappear. Creaking of the joints is often distinctly felt on movement.

MUSCULAR RHEUMATISM is a myalgia resulting from overstrain, or exposure to cold and damp. The gouty habit predisposes to it, and it is most common in men. It has no direct connection with acute rheumatism, but is probably due to inflammatory changes in the muscular fibrous tissues, associated with neuralgic affection of the nerve-endings. Its most frequent *varieties* are—

1. *Lumbago.*—The aponeurosis of the erector spinæ and latissimus dorsi is most frequently involved. The pain is often intense, and may affect the locomotion markedly. Often the onset of the pain is traced to some physical exertion, such as getting up into the saddle, or lifting heavy weights. The latter point must be borne in mind in colliery practices, for the colliers frequently demand pay on account of injury—*i.e.*, “strained back.”

2. *Pleurodynia*.—The sheaths of the pectoral muscles, intercostals, or serratus magnus, are most commonly affected. The respiratory movements of the affected side are much embarrassed. This affection may be mistaken for pleurisy, as the movements of the affected muscles often cause a distinct fremitus. The absence of other physical signs should prevent such an error.

3. *Muscular Torticollis or Stiff Neck*.—Here the cervical muscles, especially the sterno-mastoid, are affected. This condition must not be confounded with spasmodic torticollis. Many other varieties are described.

Treatment.—Keep the bowels well open by saline aperients. Turkish baths and massage, combined with the administration of iodides, form the routine treatment. The A. B. C. liniment is most useful for local application. If the condition does not speedily improve, a visit to Buxton or Bath, etc., should be insisted upon, where the patient may undergo a thorough system of massage, and course of saline waters.

ARTHRITIS DEFORMANS (rheumatoid arthritis).

Arthritis deformans is a chronic inflammatory affection of the joints, resembling gout and chronic rheumatism in many of its characters, but differing essentially from both. Although a chronic disease, the patient may have acute or subacute attacks superadded.

Ætiology.—Most common amongst females, between the ages of twenty and forty. The disease may, however, occur at any age. Heredity, phthisical history, ovaritis, grief, worry, etc., are looked upon as important ætiological factors. It is most common amongst the poor, and is favoured by exposure to cold and damp.

Pathology.—At present speculative. Possibly it is due to neurotrophic changes; and reasoning from the number of joints attacked, the symmetrical distribution of the trophic changes which lead to such grave alterations in the skin, nails,

etc., this view might seem to be the correct one. It is also possible that the disease is of microbic origin, as it is often the sequel to an acute infection, in certain cases its onset is acute, in children there is often wide-spread enlargement of lymphatic glands and of the spleen, and in some cases microbes have been found in the articular fluid. The more prevalent view is "that it is due to a settlement of micro-organisms in the affected joints, that they there produce a toxine, and that that toxine, passing into the circulation, is responsible for any nervous symptoms which occur" (Luff).

The morbid changes found in the joints affected are identical with those of Charcot's joint disease, sometimes a complication in cases of locomotor ataxia. According to Adams, the disease begins in the cartilages and synovial membranes—as splitting of the cartilage and liberation of the cells; next, absorption of the cartilage and approximation of the two articular surfaces of the joint, which by rubbing together, become very dense, hard, and highly polished; the surfaces at the same time, through pressure, become broadened out—*i.e.*, lateral expansion. Underneath the new and dense ivory-like bone, rarefaction and atrophy occur, leading to much shortening and deformity.

Meanwhile the synovial membranes become inflamed and thickened; often portions become detached, and form loose bodies in the joint. The ligaments become much thickened, and often contracted; sometimes they calcify and cause more or less ankylosis. At the margins of the joints where the pressure is less, ossification goes on, resulting in the formation of irregular bony outgrowths, termed osteophytes. The ankylosis of rheumatoid arthritis is never complete, and there is no tendency to suppuration.

Symptoms.—

The Ordinary Chronic Form.—The joints are involved symmetrically, although the process generally begins on one side, and becomes symmetrical by extension. The smaller joints, especially the metacarpo-phalangeal and inter-phalangeal joints of the hands, are usually first affected. At first the

joints may be swollen, red, and tender, but the more constant features are stiffness and gradual deformity, without any marked signs of inflammation. In confirmed cases the joint changes and the accompanying muscular atrophy cause the deformities to assume a very characteristic appearance. The lower ends of the ulna and radius project at the wrist, the metacarpo-phalangeal joints are flexed, the first phalangeal joints are over-extended, the second are flexed and the fingers deviate to the ulnar side. The joints give forth a creaking sound when moved.

When the disease is confined to the hands, the osteophytes receive the name of "Heberden's Nodosities." After the hands and wrists, the knees and ankles are most commonly attacked, but cases have been known where all the articulations of the body have become implicated. The muscular atrophy, though no doubt due largely to atrophic influences, is largely contributed to by non-use.

Besides Heberden's nodosities, a monoarticular form of rheumatoid arthritis has been described. This type differs from the polyarticular form, *by affecting the spine or hip-joint, in elderly men.*

An Acute Form of the disease is known, but is much less common. After a febrile onset, the joints become distended with fluid, and are speedily disorganised. Erosion of the cartilages with grating follows, but there are no osteophytes. This may subside in about a month, or become chronic. In children the joints are less disorganised.

Diagnosis.—From Charcot's joint, by the history and absence of ataxic symptoms. From gout, by the absence of tophi, no excess of uric acid, etc.

Treatment.—No treatment can cure this disease, except in its earliest stage, but it is often arrested by careful attention to the general health. Liberal diet, fresh air, and gentle exercise, avoiding pain, and a dry warm climate are beneficial. Of drugs cod-liver oil, iron, and arsenic are chiefly used. Garrod recommends iodide of iron, and Luff carbonate of

guaiacol (gr. v-x thrice daily). Massage is often of great service. At first light general massage only should be employed, but after a few days frictions may be made over the affected joints. Passive movement is also useful.

DIABETES MELLITUS.

A disease characterised by an increased discharge of pale urine of high specific gravity, containing a quantity of grape sugar. It is attended with great debility and progressive emaciation. To constitute diabetes, the sugar must be *permanently* present. A temporary glycosuria is not diabetes.

Ætiology. Many cases are distinctly hereditary. Men are more often affected than women. Jews especially seem prone to this affection. It is rarely seen amongst children. The principal exciting causes are—

1. Tumours (especially gummata) affecting the fourth ventricle.
2. Diseases of the spinal cord (occasionally).
3. Mental disturbances, such as worry, grief, fright, etc.
4. The gouty state.

Lately much attention has been drawn to the fact that obese persons frequently suffer from diabetes; but it is quite unsettled to what extent the “fatty habit” may play the part of an ætiological factor.

Morbid Anatomy.—

1. *The Nervous System* shows no constant changes. In many cases, tumours or degenerative changes in the floor of the fourth ventricle, in the medulla, or at the base of the brain, and sclerosis of various sympathetic fibres have been found.

2. *The Pancreas.*—Is sometimes atrophied, fibrosed, and on section shows extensive areas of fatty degeneration, and fat necrosis. The islands of Langerhans, which are supposed to furnish the internal secretion of the pancreas, have been found in a state of hyaline degeneration.

3. *The Liver*.—Is often cirrhotic and peculiarly pigmented, the changes, however, are by no means constant. The organ may be hypertrophied.

4. *The Kidneys* are usually cirrhotic, though not extensively so; hyaline degeneration of the tubules is usually most marked.

5. *The Blood* shows four remarkable changes—

(1) *The presence of polynuclear leucocytes containing much glycogen.*

(2) *An excessive amount of sugar in the plasma.*

(3) *The presence of much fat; which may form a creamy layer on the clotted blood.*

(4) *A reduction in the alkalinity of the blood.*

The Urine.—Both quantity and specific gravity are increased, the former to the extent of ten or even twenty pints per diem—the latter from 1025 to 1045 or more. It is pale in colour, clear, and acid in reaction. The quantity of sugar ranges from two to forty grains per ounce, or twenty ounces and upwards daily. Uric acid is not increased, but both *urea* and *phosphates* are. Albuminuria is often present in the late stages. Amongst other constituents which must be remembered are β -oxybutyric acid (often present in the late stages), and diacetic acid. The relations of these products to diabetes are still undetermined. *Outside the body*, glucose can, by taking up oxygen, be transformed into β -oxybutyric acid, with an intermediate stage of aldehyde. *A very tempting theory is to assume that such a change takes place in diabetes.*

Diacetic acid is also important, for it has often been confused with and mistaken for acetone, which is frequently present. This is of the utmost importance, for “Acetonuria” has been held responsible for causing the well-known coma of diabetes. It has, however, been conclusively shown by von Jaksch and others, that *acetone* cannot be the *cause* of the coma; yet even von Jaksch admits that when the acetone reaction appears in the urine, it usually heralds the onset of the coma and death. Possibly the explanation is that diacetic acid undergoes the following changes in the alkaline plasma. It first combines with

the soda in the blood ; then splits up into acetone, alcohol, and the bicarbonate of soda— $C_6H_{12}NaO_3 + 2H_2O = C_3H_6O + C_2H_5O + NaHCO_3$. This change would account for the diminished alkalinity of the blood, and *possibly* the alcohol so formed may account for the coma. But the coma is held by some to be due to fat embolism, or the circulation of fatty acids. The most generally held view is that it is due to acid-poisoning caused by the presence of large quantities of β -oxybutyric acid in the blood.

Pathology.—When we come to discuss the real causation of diabetes, we are beset with great difficulties ; in fact we are not even out of the realm of speculation, in spite of much hard honest labour expended on the subject. The starting-point of the disease is probably not always the same, and many cases show at the autopsy no lesions of brain, liver, or pancreas, such as might be supposed to cause the disease.

Normally about 0·1 per cent of sugar is present in the blood, and a minute trace, which the clinical tests cannot detect, in the urine. When the amount in the blood is more than 0·2 per cent an appreciable glycosuria occurs. Even in health glycosuria may be produced by an ingestion of sugar beyond the physiological limit. This, however, is temporary.

The appearance of sugar in the urine, then, is due to excess of sugar in the blood, and this in turn may be due either to (1) *excessive production* of sugar in the organism, or to (2) *diminished destruction*. The usual view of the glycolytic function of the liver is that the organ converts the carbohydrates brought to it from the intestines into glycogen. This is stored up in the liver and the muscles as a reserve. It is gradually reconverted into sugar according to the needs of the organism, and carried by the blood to the tissues, where it is oxidised. Pavy, however, holds that the liver does not supply sugar to the blood, but removes it *from* it, and converts it into fat. There is, therefore, a little sugar in normal blood because the liver fails to remove it all, not because the liver is the source of sugar-supply.

In mild forms of the disease a diet free from carbohydrates

stops the glycosuria. The sugar is therefore derived from the carbohydrates of the food. According to Seegen's view the liver cells are unable to transform the carbohydrates in a normal fashion. In Payy's view these cells do not destroy the sugar brought to them, and excess of it gets into the circulation and thence into the urine. It is also possible that there is an excessive transformation of glycogen into sugar. In severe forms glycosuria persists though all carbohydrates are stopped, and even though no food is taken. In these cases sugar may be formed from proteids and from fat.

The causes leading to this excess of sugar in the blood have already been mentioned. In not a few cases change in the *central nervous system* is associated with the origin of the disease. It may be functional, as the result of shock or fright, or organic, as the result of tumours or degenerations; and the absence of recognisable change, even by the microscope, does not preclude the possibility of minute changes in the nerve cells.

As regards the *pancreas*, its total extirpation in animals is followed by diabetes, and very often in man extensive changes (cancer, cirrhosis, atrophy, fatty degeneration) have been found post mortem. In other cases no macroscopic or microscopic changes have been found.

Bunge and others have suggested that in some cases diabetes may be caused by pathological chemical changes in the muscles.

Symptoms.—*Two* forms of diabetes exist—viz., the acute and the chronic forms. In the acute form, the symptoms assume a grave type very rapidly, and post mortem in such cases the pancreas has been found to be extensively diseased. In the chronic form, on the other hand, the symptoms are often obscure, and beyond the peculiar characters of the urine and attacks of dyspepsia, the patient may for a long time maintain a fair standard of health.

Taking a typical case of diabetes, the characteristic symptoms are, the passage of large quantities of pale urine, great thirst, voracious appetite, progressive muscular weakness, a dry, parchment-like sallow skin, emaciation, and the development of

some of the complications tabulated below. In the later stages the pulse becomes very feeble, severe attacks of diabetic dyspnœa, or "air hunger," add to the general misery, the breath gives out a peculiar sweetish apple-like odour, and only too frequently coma ends the scene suddenly. Often, however, the patient is carried off by a critical diarrhœa, pneumonia, etc.

In chronic diabetes some of the more common complications require special mention. They are—

1. *Cutaneous Lesions*.—Boils, carbuncle, eczema, especially of the vulva in women, and accompanied by intolerable itching.

2. *Visual Changes*.—Retinitis, soft cataract, and more or less optic neuritis. Retinitis and neuritis are occasional complications; cataract is common.

3. *Nervous Changes*.—The knee-jerk is often absent, especially in severe cases. This is due to peripheral neuritis, which is manifested by cramps in the legs, tingling, numbness, or neuralgic pains. There is loss of sexual desire. The nervous changes may lead to—

4. *Atrophic Changes*.—Gangrene, especially perforating ulcer of the foot, brittleness of the nails, etc.

5. *Renal Changes*.—Albuminuria, at first without Bright's disease, later with more or less cirrhosis of the kidney and symptoms arising from such a condition.

6. Tubercular affections of the lungs are very common.

Diabetic coma may arise suddenly, or may be preceded by headache, restlessness, and a feeble, rapid pulse. It is favoured by excitement or fatigue. The temperature is subnormal, there is drowsiness, and the respirations are prolonged and sighing. There is an odour like that of apples in the breath and urine. The urine may contain acetone, diacetic acid, and β -oxybutyric acid. The drowsiness deepens to complete coma, and ends in death, usually without convulsions.

In addition to the above common complications, there are often grave mental changes. A peculiar form of high-stepping ataxia has been described by some authors. It should not be

forgotten that in diabetes diarrhœa may be easily provoked, and I have seen two deaths from this cause, following the administration of simple purges. The dyspnœa is distinctly peculiar, for it is *rarely accompanied by cyanosis* and has received the name of "air hunger."

Diagnosis.—The urine properly tested at frequent intervals forms the best means of diagnosis (see Examination of Urine). Remember the wasting of muscle may be concealed by obesity.

Prognosis.—In confirmed cases the outlook is bad. In the more chronic varieties the disease may be arrested for a considerable time. Some patients have had diabetes for twenty years. Cases occurring in later life are apt to run a chronic course. The younger the patient the more likely and the more rapid is a fatal issue.

Treatment.—Diabetic patients often eat enormously, and it must be at once apparent how difficult it is to balance the economy under such conditions, for if we limit the *carbohydrates* we must increase the *proteids*, and thus *throw an enormous amount of work upon the already handicapped liver and kidneys*. If physiology be correct, to give an excessive amount of proteids is certain to prove in time disastrous to the liver, and hence such practice is to try to cure diabetes by doubtful means. *But all authorities agree that the use of carbohydrates must be restricted to a minimum.* Fatty diet has been suggested as a compromise; but there is already an excessive amount of fat in the blood in most cases, and further it must be remembered that the tissues have the power of building up fat from the fatty acids which must be formed during excessive decomposition of proteids; still some eminent authorities advise a more or less fatty diet. Sir William Roberts's dietary is appended as a guide.

ALLOW

Butcher's meat, poultry, game and fish. Cheese, eggs, butter, fat and oil. Broths, soups, and jellies made without meat or sugar. Cabbage, endive, spinach, broccoli, lettuce,

spring-onions, watercress, celery. Dry sherry, claret, brandy, and whisky. Tea, coffee (without sugar), soda-water, bitartrate of potash water.

FORBID

All saccharine and farinaceous food, *bread*, potatoes, rice, tapioca, sago, arrowroot, macaroni; turnips, carrots, parsnips, beans and peas.

Liver contains much sugar-forming substance, therefore, oysters, cockles, and mussels, which contain enormous livers, are forbidden.

All sweet fruits. All sweet wines.

It will be seen from the above table that there is a pretty extensive range or choice of food, but unfortunately the two great drawbacks are the non-allowance of bread and potatoes. The substitutes for bread are either nauseous or too expensive for general use. The principal substitutes are almond biscuits, gluten bread, charred bread, soya bread, etc.

The diet must not be abruptly altered, but injurious substances must be gradually removed.

Donkin's treatment, by an exclusive diet of skimmed milk, does not usually act well, except in obese people.

Medicinal Treatment.—Codeine and morphine seem to be the most beneficial drugs. Antipyrin is credited with the power of reducing the amount of sugar, if given in doses of ten grains thrice daily. Saccharine and glycerine are no good as curative agents, but they may be used for sweetening purposes. Arsenic and strychnine are useful tonics. Massage, electricity, cod-liver oil, etc., are all useful in various cases. The great thirst may be relieved by the administration of large doses of citrate of potassium freely diluted. The complications must be treated on those principles laid down under their respective headings.

DIABETES INSIPIDUS

Is a rare condition in which an excessive quantity of pale limpid urine is secreted, free from sugar, albumin, and other abnormal constituents; and accompanied by insatiable thirst.

Ætiology.—The disease is commonest in the young, and attacks males oftener than females. Bernard produced polyuria by puncturing the fourth ventricle higher than the centre for producing saccharine urine; and it has been suggested that diabetes insipidus is due to some disturbance of this centre, but it is probable that the condition is due to a vasomotor paralysis of the renal blood-vessels, produced either by local causes in the abdomen, or by central disturbance. It has sometimes been produced by severe mental shock. The temporary polyuria after a hysterical fit is well known.

Morbid Anatomy.—Various conditions have been found, of which the most frequent are the natural results of polyuria—hypertrophy of the bladder and dilatation of ureters and renal pelvis. None are constant.

Symptoms.—Polyuria, the watery constituents alone being increased; as much as 15 to 40 pints of this pale urine may be voided in the twenty-four hours. The specific gravity is very low, from 1008 down even to 1001. Dyspepsia, great thirst, mental irritability, and muscular weakness are also frequently prominent symptoms. Though the disease is rarely fatal, the persistent thirst and frequent micturition prevent sound sleep, and may undermine the general health.

Treatment.—Valerianates, iron, strychnine, galvanism, and the bromides have all been used. The valerianates must be given in large doses. It is wrong to try to limit the amount of fluid swallowed. There is no evidence that any drug materially affects the course of the disease.

Diagnosis.—Care must be taken not to mistake this condition for the polyuria of Bright's disease, etc. Note the absence of casts, and the low sp. gr. Hysterical polyuria is transitory, not permanent.

DISEASES OF THE ALIMENTARY TRACT.

1. THE MOUTH.

THE mouth is liable to a number of diseases, chiefly inflammatory, which may be local or symptomatic of general disease. The local diseases are mainly due to organisms ingested or inhaled, but may also be produced by the coughing up of infected sputum, as in phthisis. Pathogenic organisms may be present even in health and require a suitably modified soil before they can cause disease. The most frequent are the *Staphylococcus pyogenes aureus* and *albus*, the *Streptococcus pyogenes*, the *Diplococcus pneumoniae*, and the *Micrococcus tetragenus*. Fungi, such as the *oidium albicans* and *monilia candida*, are also found.

THRUSH (PARASITIC OR APHTHOUS STOMATITIS)

Is a *specific* disease dependent on the development and multiplication of a special fungus termed the *oidium albicans*; it is frequently met with in weakly children and infants.

The fungus is probably identical with the *Mycoderma vini*, and is therefore a mould. It causes the formation of milk-white or greyish adherent patches on the mucous membranes of the mouth and pharynx of the child attacked.

Microscopically these patches consist of epithelium united into a membrane by twisted filaments (the fungus), which are often branched and composed of long cells joined end to end,

and constricted at the joints. Both hyphæ and spores are to be seen.

The patches may extend, though rarely, to the œsophagus, stomach, and cæcum.

Symptoms.—The mouth is *dry*, and tender or painful; there is usually much debility and gastric disturbance. Small white roundish patches form, and may coalesce, producing larger areas. The patches, at first adherent, are later easily detached, leaving behind them little ulcerations.

Treatment.—Absolute cleanliness of the feeding bottles, the avoidance of stale milk, and the frequent swabbing out of the mouth with a solution of boracic acid or permanganate of potash, are the principal indications for successful treatment. Everything must be done to improve the debilitated condition generally.

STOMATITIS.

An inflamed condition of the mouth is common during dentition, certain fevers, and morbid states of the blood. It may be also caused by mechanical irritation or by the internal use of mercury.

Such a condition, however caused, is likely to be attended with the following symptoms:—

1. *Pain and difficulty* in mastication, articulation, and deglutition, in proportion to the severity of the disease.

2. Increased salivation.

3. Fœtor of breath.

4. Constitutional disturbances.

Like all inflammations of mucous membranes, the condition varies much in severity, and accordingly the following types are described:—

The simple or catarrhal stomatitis, a mild form which readily yields to the local application of glycerine and borax. In acute cases the surface is very red and raw, in milder cases irregular white patches show increased production of epithelium.

Ulcerative stomatitis, or putrid sore mouth, characterised by the formation of small vesicles or yellowish patches of a diphtheroid nature. The vesicles after rupturing leave small greyish ulcers. They appear on the gums, tongue, lips, etc., but seldom upon the pharynx; they spread by contact. They may extend deeply, exposing the alveolus.

This type has assumed an epidemic form in localities where cattle affected with foot-and-mouth disease were present. The relations between the two conditions have, however, not yet been determined.

Treatment.—Touch the ulcers with 10 per cent solution of nitrate of silver. Improve the general health of the patient with tonics. A mixture of hydrochloric acid, chlorate of potash, and glycerine, freely swabbed over the mouth, often acts like magic.

NOTE.—True ulcerative stomatitis is more common *after* the period of dentition.

CANCERUM ORIS (NOMA).

This is a form of gangrenous stomatitis, happily now rare, but sometimes seen in debilitated children between the ages of two and five years.

Ætiology.—The disease follows upon defective nutrition and bad hygiene. It is often a sequela of the acute infections of childhood, especially measles.

Pathology.—At first there appears a small diphtheroid patch of necrosed tissue with a general inflammation around it. This inflammatory zone extends and becomes brawny in character, the slough separates, and the ulceration goes on until the cheek is perforated. The disease frequently kills the patient before necrosis is well marked, but sometimes the gangrene extends to the jaw, malar bones, tongue, etc., before a fatal termination is brought about.

Symptoms.—The constitutional disturbance is very great, although fever is moderate, and usually the typical typhoid state rapidly ushers in a fatal ending.

Treatment.—Highly unsatisfactory, for it has been fairly well demonstrated that the virulence of the affection is not dependent on the poison as much as on the peculiar debilitated state of the patient, and the unhealthy surroundings in which he exists.

Complete excision of the diseased area by the knife, with the administration of quinine and stimulants are the best measures. To parts which cannot be excised, nitric acid or pure phenol should be freely applied. Should the child recover, a plastic operation will be necessary later on.

ACUTE TONSILLITIS.

Inflammation of the tonsils is not only a common complication or symptom of many fevers, but is also a common primary affection; when the inflammatory process goes on to suppuration (as it frequently does) it is popularly termed quinsy.

Ætiology.—Acute tonsillitis occurs most frequently in young people, but may occur at any age. It is often associated with, or precedes, acute rheumatism. Primary tonsillitis is a direct infection. In either case the organisms found are mainly the pus-forming cocci.

Symptoms.—The onset is sudden. The throat is hot and dry, and the temperature runs up rapidly to 104° or more, accompanied with severe frontal headache. The tongue is foul and the breath fœtid. The glands below the jaw are enlarged, and surrounded by diffuse tender swelling. There is great pain on movement of the jaw or in swallowing. The tonsils are enlarged and congested, as are the fauces and soft palate. A thin dirty yellow exudate may cover the surface of the tonsils. In some cases it may be thicker, patchy, and semi-membranous in appearance, differing however from diphtheritic membrane in that it strips off readily on brushing with 10 per cent solution of silver nitrate.

If treatment is effective, the acute symptoms subside in four or five days, and the patient is well in about a fortnight.

Sometimes, however, suppuration occurs in the substance of the tonsil, and an abscess forms on one or both sides (*quinsy*). In such a case the fever does not subside, the swelling becomes so great that the mouth can hardly be opened, and fluctuation can be felt by the finger on one or other side. The abscess may rupture spontaneously into the mouth, and sometimes it leads to profuse hæmorrhage.

Diagnosis.—The difficulty lies chiefly in distinguishing between acute tonsillitis and diphtheria. There may be membrane in the former; it may be absent in mild cases of the latter. It is often impossible to decide on a first examination whether diphtheria is present. Even a film prepared direct from a swab may not show the characteristic bacilli. Cultures made upon blood-serum clinch the diagnosis of diphtheria, but time is lost in this examination, as the colonies require about eighteen hours for their development. It is always safest in doubtful cases to inject antitoxin at once. If the case is afterwards proved not to be diphtheria, no harm is done.

Treatment.—Begin by opening the bowels freely with calomel followed by a saline, and keep them freely open. Subdue fever and relieve headache by antipyretics or salicylates. The salicylates (gr. x-xv every three hours) have also an excellent effect on the general course of the disease. Locally use glycerine of belladonna and hot fomentations externally. Paint the throat before food with solution of cocaine (gr. v-x to the ounce), or in mild cases gargle with Potass. Chlor. gr. lxxx, Acid. Hydrochlor. dil. ʒiiss, Glycerini ʒiv, Aq. ad ʒviij. Antiseptic sprays are also useful.

The abscess of quinsy should be opened with a guarded knife. In the *low subacute* forms, frequently seen in overworked patients, nothing succeeds better than a good swabbing once for all of the liq. ferri perchlor. fort. It is not a pleasant application, but is very efficacious. Nourishing diet and tonics are absolutely required during convalescence.

II. THE ŒSOPHAGUS.

ŒSOPHAGITIS.

Inflammation of the œsophagus may be acute or chronic. The acute form arises (1) from scalds or burns, or the swallowing of corrosive poisons; (2) as a complication of the specific fevers; (3) towards the end of wasting diseases; (4) in infants, as a purely catarrhal type, often without apparent causes. Ulceration or cicatricial stricture may follow upon acute œsophagitis.

The **symptoms** are, mainly, pain in swallowing, tenderness over the cervical portion, and spitting of mucus.

Treatment consists in the administration of bland fluid food. Where corrosive poisons have been swallowed, rectal feeding must be instituted.

CHRONIC ŒSOPHAGITIS may follow the acute affection, may arise above a stricture, or may be the result of chronic alcoholism.

It tends to lead to ulceration and perforation. In alcoholics, morning vomiting, usually of œsophageal mucus, but sometimes mixed with the contents of the stomach, is the principal sign. Where the vomit comes only from the œsophagus, its reaction is alkaline; if the gastric contents are also expelled, the reaction is acid.

STRICTURE.

Simple stricture of the œsophagus may be (1) cicatricial, (2) spasmodic (œsophagismus).

CICATRICIAL STRICTURE is usually due to the after effects of burns, scalds, or corrosive poisons; but it may follow upon cicatrization of ulcers of whatever kind, or upon external pressure, as in aneurism or mediastinal tumour.

Obviously, the stricture must vary in its length with the cause that has produced it. In different cases the gullet may be slightly narrowed, or so much that even fluids can scarcely pass.

The **symptoms** depend to some extent on the position of the stricture. Dysphagia is common to all sites. If the stricture is high in the tube, the return of food may be immediate, if low, after a slightly longer interval. Emaciation follows when the narrowing is great.

The position of the stricture, and the degree of stricture, must be determined by the passage of an œsophageal bougie, after all serious intrathoracic diseases have been carefully excluded. Aneurism, mediastinal tumour, valvular affections of the heart, etc., preclude such an examination.

Treatment.—Progressive dilatation; if the stricture is impassable, gastrostomy.

SPASMODIC STRICTURE occurs in neurotic individuals, especially young women. It commences suddenly, usually during a meal, the food sticking for some time, and then either passing on to the stomach or being returned. It is attended by severe burning pain and retching. There is little emaciation.

On passing the sound, the stricture will be found to occupy varying positions at different examinations.

Treatment.—Pass a full-sized bougie, twice or thrice weekly. This is often sufficient to cure the condition. At the same time treat any nervous disturbance that may be present.

TUMOURS.

Innocent tumours occasionally occur, but are of small importance. Sarcoma is extremely rare.

CANCER, of the type of squamous-celled epithelioma, is common between the ages of 45 and 60, and most frequent in the male. The growth is annular, infiltrating all the coats, and ulcerates rapidly from the passage of food over it.

The **symptoms** are increasing dysphagia, regurgitation of food mixed with mucus and blood, and rapid emaciation. The passage of a bougie locates the site of the stricture. This must be carefully done, as the diseased wall is very liable to perforation.

Death occurs from six months to a year after the onset of symptoms. It may be due to starvation, or to complications due to direct extension of the growth (septic broncho-pneumonia, perforation of the aorta, etc.). Metastases are uncommon, except in the thoracic and cervical glands.

Treatment.—Where the cervical part is affected, excision of the tumour. In other cases permanent intubation, or if this is not tolerated, gastrostomy.

III. THE STOMACH.

EXAMINATION OF THE STOMACH CONTENTS.

In order to have a clear idea of the various disorders this viscus is subjected to, it will be necessary to recall the main facts relative to the digestive processes that go on in the stomach. We know that starchy foods become converted into maltose, with an intermediate stage of dextrines, owing to the continued action of the ptyalin derived from the saliva, which is not inhibited by acid during the first half hour; proteids into peptones, with an intermediate stage of albumoses, owing to the direct action of the gastric juice; and that fats are also acted upon to a slight extent, inasmuch as the proteid envelopes of the fat cells are dissolved. The caseinogen of milk is also converted into coagulated casein by the action of rennin. We can easily understand, then, that such digestive processes can be retarded by—

1. Putting into the stomach a larger amount of food than it is capable of digesting.
2. Deficiency either in quantity or quality of gastric secretion.
3. Deficient movements of the stomach, through muscular atony.

It is highly necessary, then, to know the exact state of secretion, and the amount of motor or peristaltic power.

During the process of digestion the stomach contents at various intervals give three distinct reactions—

1. Immediately after food is taken they are faintly alkaline from the saliva swallowed.
2. The next half-hour they are slightly acid from lactic acid.
3. After this time they gradually become acid from the presence of free HCl.

The first acid to appear as a rule is lactic acid, and it must be remembered that this acid is a perfectly normal constituent during the first half-hour or so of digestion; after *an hour there should only be the faintest traces*. It must not be forgotten also, that HCl is hostile to the action of bacteria; consequently if lactic acid is found in excess two hours after a standard meal we can at once conclude that HCl is deficient.

When it is desired to ascertain whether gastric digestion is normal, a test meal is first given; then a portion of the gastric contents is pumped out, after a definite interval, through an elastic œsophageal tube, and chemically examined.

Examination by this means is chiefly useful in obscure cases of gastric neurosis, and functional disorder where there is no vomiting, and where, in an early stage, carcinoma is suspected, but the symptoms are not diagnostic.

Conditions contra-indicating the passage of the gastric tube are the same as those that contra-indicate sounding the œsophagus, with in addition the presence of acute gastritis, gastric ulcer, or a late stage of carcinoma. Where vomiting is present, examination of the vomited matters may suffice.

The first step is to determine the *total acidity* of the gastric juice by a decinormal solution of NaHO, of which 1 cc. = 0.00365 grm., HCl. 10 cc. of the juice are placed in a porcelain capsule with a few drops of a 1 per cent solution of phenolphthalein, and the NaHO is added drop by drop. A pink colour forms, which disappears on shaking, but becomes permanent when neutralisation is complete. The percentage acidity can be calculated from the number of cc. of NaHO

employed, but it is usual to express the acidity simply in terms of NaHO. If 6·5 cc. are required for 10 cc. of juice, 65 would be required for 100, and the acidity of the juice is spoken of as 65.

We next determine whether the acid is present as free acid or in combination, by adding to the filtered juice a solution of Congo red. In the presence of free acid a dark-blue colour is produced.

To determine that the free acid is HCl, a series of colour tests is used.

1. Dimethyl-amido-azo-benzol (0·5 per cent alcoholic solution) gives a cherry-red colour with the unfiltered juice in the presence of free HCl.
2. Gunsberg's test (alcoholic solution of phloroglucin and vanillin). If this be evaporated with a few drops of the juice, a rosy tint appears in the residue.
3. Boas' test (alcoholic solution of resorcin) is carried out in the same way.

It is unnecessary here to give the details of the quantitative estimation of the total amount of free hydrochloric acid.

Uffelmann's Test for lactic acid is a most convenient one for general practice. The reagent is made by colouring (violet) a 1 per cent solution of carbolic acid by the addition of tinct. ferri perchlor.

METHOD.—Take filtered gastric contents, add an equal quantity of the reagent.

If lactic acid be present—canary yellow.

If butyric acid be present—dirty grey.

Tests for Peptone—

Characters.—

1. Like all proteids it is—
 - (1) Insoluble in strong alcohol.
 - (2) Gives xanthoproteic reaction.
 - (3) Gives a pink colour reaction with copper sulphate.

2. Unlike all other proteids peptone is diffusible.
3. Heat does not coagulate peptone (*unlike albumin*).
4. Nitric acid does not precipitate peptone (*unlike albumin*).
5. Pure neutral ammonium sulphate does not precipitate peptone, while it precipitates albumin, globulin, and albumose.

Albumose or Propeptone is intermediate between albumin and peptone. It gives the typical reactions of proteids. Like albumin it is non-diffusible. Like albumin it is precipitated by nitric acid in the cold, but the precipitate *dissolves on heating and returns on cooling*. Neutral ammon. sulphate precipitates it as it does albumin.

Rennin is the milk-curdling ferment of the gastric juice. Pancreatic juice contains a similar ferment.

The curdling of milk is due to the coagulation of the alkali-albumin casein. Any acid will curdle milk, but rennin does something more than mere curdling—it renders the curd more digestible. It is a remarkable fact that rennin fails to “curdle” milk if lime salts are not present.

Presence of Pepsin.—The presence of pepsin can be inferred by the following process:—Take a filtered solution of the gastric contents, add to it 0·2 per cent of HCl; place in this solution boiled fibrin—the fibrin will be dissolved as in normal gastric juice.

Vomited Matter may be tested, instead of drawing off the contents of the stomach by the œsophageal bougie, but the results are likely in such cases to lead to erroneous opinions.

Power of Absorption can be tested (so it is said) by observing the rapidity with which KI (which has been swallowed in a gelatine capsule) appears in the saliva.

Motor Power of the Stomach is estimated by testing with ferric chloride for salicyluric acid in the urine at various intervals after a certain quantity of salol has been taken by the mouth. Salol should not be decomposed by the stomach contents, which are acid, but only when it meets with an alkaline fluid—*i.e.*, in the small intestines. If the

urinary reaction (a violet colour) is delayed beyond an hour and a half, the motor power of the stomach is held to be deficient.

ACUTE GASTRITIS.

Acute gastritis may be catarrhal, toxic, or infective. The catarrhal form is due to the severe irritation set up by the ingestion of unripe fruits, decomposed tinned meats, shell-fish, etc. It may also follow upon the abuse of alcohol or even of tea. Persistent hyperacidity (especially when due to organic acids), and delay of food in the stomach, favour its production. It is frequently a complication of convalescence from febrile diseases, low inflammation, etc.

Pathology.—The mucous membranes show the usual hyperæmia, exudation, and increased mucous secretion observed when mucous membranes are inflamed. The various epithelial cells of the numerous glands may become highly granular, undergo mucoid degeneration or desquamate. There may be minute extravasations of blood, hæmorrhagic erosions, pustules or aphthous patches.

The submucous coat is infiltrated, and the whole stomach wall may be congested. The pyloric region is principally affected.

Symptoms.—Sudden onset, severe epigastric pain passing through to the back, and accompanied by deep *diffuse* tenderness; vomiting of food mixed with abundant mucus or streaked with blood; absence of free HCl; slight fever, marked prostration.

Treatment.—The first point is to secure absolute rest for the stomach. It should be washed out once or twice at the outset with a dilute alkaline solution (NaHO 1 - 1000). This removes irritants, and often stops vomiting. If retching continues give morphia hypodermically, and for pain apply belladonna fomentations. For thirst small pieces of ice may

be sucked. Feed for the first twenty-four to thirty-six hours by nutrient enemata and suppositories, and return to gastric feeding very gradually and carefully.

CHRONIC GASTRITIS (chronic catarrh)

may follow the acute form, or may be due to prolonged gastric irritation. In the latter case there is hyperacidity in the early stages; in both forms there is later hypochlorhydria. There is lessened motor activity, often leading to dilatation and bacterial fermentation. Degenerative changes, especially fatty, affect the glandular cells. Mucus is at first continuously secreted; later, if the mucosa is widely destroyed, the secretion may be lessened. Fibrosis occurs between the glands, and the contracting fibrous tissue narrows their necks, and thus causes intra-glandular cysts. These can be seen chiefly in the pyloric region, forming little pin-head-like projections on the surface of the mucosa (*état mamellonné*).

Symptoms.—The course is protracted, afebrile, and marked by subacute exacerbations. Pain comes on at once or soon after food, with diffuse epigastric tenderness greater in certain spots (supposed to mark the site of erosions) than elsewhere. Nausea is frequent, vomiting less so, though there is always vomiting at some periods of the course. It may be related to food, or occur in the morning. The vomit contains much mucus, little HCl or pepsin, and partly digested food. Constipation and flatulence are present, the tongue is thickly furred, and there is some wasting.

Treatment.—Rest to the whole organism, and especially to the stomach. Begin by washing out. Give at first only fluids in small quantities and often, and increase the diet very carefully. Correct constipation. For drugs, dilute mineral acids after food, combined, if there is much pain, with Acid. Hydrocyan. dil. or Lig. Morph. Hydrochlor. It is only in the earlier stages that cure can be expected, and then only if faulty dietetic habits are permanently abandoned.

Toxic Gastritis arises from the ingestion of corrosive or irritant poisons, or poisonous foods. See works on toxicology.

Infective Gastritis is usually secondary to a general infection. False membrane may be found in diphtheria, pustules in small-pox, multiple abscesses in pyæmia, etc. The condition is generally diagnosed *post mortem*.

ULCER OF THE STOMACH.

By this lesion is meant a form of ulcer, which is peculiar to the stomach, the first part of the duodenum, and (according to some authorities) the lower end of the œsophagus. Though usually single, there may be more than one ulcer present.

Site.—Solitary ulcers are most frequently situated on the posterior wall, near to, or involving the lesser curvature. Sometimes they are observed near the pylorus, but rarely do they attack the greater curvature. Occasionally they are situated on the anterior wall, and then are very liable to perforate.

Characters.—The ulcer may be acute or chronic, the acute form being small and sharply punched out, the chronic larger, funnel-shaped (broader at the level of the mucosa than in the deeper coats), and with thickened edges. The shape is circular or oval. The edges are *not* undermined, and there is an absence of vascularity in the margins and base. The floor is formed by the submucosa, the muscular coat, or the serous coat; the latter is not unfrequently thickened, and adherent to the neighbouring organs. These adhesions are of the utmost importance, and go far to explain why perforation is not more common in cases of gastric ulcer. As the ulcer deepens, its floor or base becomes narrower, so that the walls come to have a terraced appearance, and an oblique direction. If perforation occurs, the aperture in the serous coat is of small size, but presents the same clean-cut, punched-out appearance, that distinguishes the margins. Perforation is frequent in

acute ulcer, but may occur in either form. In the healing of the ulcer, if the mucosa be alone involved, the granulation tissue develops from the edges and the floor, and the newly formed tissue gradually contracts and unites the margins, leaving a smooth scar.

In larger ulcers which have become deep, and involved the muscular coat, the cicatricial contraction may cause serious changes, the most important of which is, narrowing of the pyloric orifice and dilatation of the stomach. A common result of the ulcerative process is perforation of the gastric walls, and consequent fatal peritonitis. On the other hand, it is not uncommon for adhesions to form between the walls of the stomach and neighbouring organs, and the ulcer, after perforating the gastric walls, burrows into the pancreas, spleen, or liver. Large vessels may be eaten into in a similar manner, and bring about a fatal issue from hæmorrhage. Other complications through the ulcerating process are —

1. Perforation into the pleura.
2. Gastro-duodenal fistula.
3. Perforation into the lesser peritoneum, giving rise to subphrenic abscess.

Pathology.—Nearly all observers agree that the ulcer is due to two immediate factors—

1. A devitalisation of certain areas of the gastric mucous membrane.
2. Erosion of these areas through either the digestive action of the gastric juice, or the mechanical irritation of indigestible food.

Probably in all cases many factors are present, and I think the best way of summing up is as follows: deficient or indifferent quality of blood supply to the walls of the stomach tends to cause—

1. Alterations in its secretions.
2. Local thrombosis of the nutrient arteries.
3. Deficient movement or peristalsis of the viscus.

Thus we may get hyperacidity of the gastric juice ; certain areas of the stomach are rendered very vulnerable through the local thrombosis, and lastly the defective peristalsis favours the concentration of the irritating gastric contents at certain points. Consequently there can be no difficulty in understanding how ulceration of the mucous membrane comes about in the thrombosed areas. If we grant this, then we must allow that such a down-grade process must be powerfully assisted by irritating food, and anything which tends to diminish the alkalinity of the blood ; for it must be remembered that physiologists hold that the alkalinity of the blood normally plays a part in preventing the stomach from digesting itself.

The theory of thrombosis is due to Virchow, but it does not explain all the cases. Ulcer is very common in chlorosis, and one does not in that disease find thrombosis occurring in other parts. Where ulcer is connected with septic states, embolism is no doubt the cause. In other cases Sidney Martin suggests bacterial invasion. If the gastric activity is lessened, bacteria may persist, enter the pyloric glands (where HCl is not secreted), and grow there and in the submucosa.

Ætiology.—Gastric ulcer is most commonly met with in young women, especially those who lead a sedentary life, who live in areas shut out from the sunlight, or who are the subjects of chlorosis or anæmia. It is much more frequent in women than in men, in the proportion of about 5 to 1. In women it is a disease of early adult life, in men it becomes more frequent towards middle age. Occupation has no very marked effect in predisposing to it. Extensive burns may be followed by gastric or duodenal ulcer. Multiple ulcers are not uncommon in septic conditions.

Symptoms.—No disease or condition may have on the one hand more characteristic, or on the other hand more ill-defined symptoms than gastric ulcer. Taking a typical case, we may expect—

1. Pain and tenderness over the epigastric region. The pain is severe and shoots through to the back (xiphi-vertebral),

is rendered worse by eating, or by firm pressure. The tenderness is *very localised*.

2. Vomiting after meals; the food usually returns quickly, and this often gives much temporary relief.

3. Hæmatemesis may be very copious, and occurs probably in quite half the cases. It may be directly fatal, or comparatively slight. Melæna is present in about 10 per cent of cases.

4. The appetite remains good, but the patient is afraid to eat, lest she set up pain.

5. The tongue is clean, and may be pale and flabby. There is little wasting.

6. There is generally excess of free HCl in the stomach contents (hyper-chlorhydria). Increase of the organic acids is rare.

These symptoms in combination with the history, anæmia, and the absence of a tumour, point strongly to the presence of an ulcer. I must, however, warn beginners not always to expect such typical signs. Often, indeed, the symptoms are very slight, and a copious or even fatal hæmorrhage, or the occurrence of perforation, may be the first indication of such a condition.

Bucquoy emphasises the following symptoms as diagnostic of the duodenal ulcer—

1. Sudden intestinal hæmorrhage (melæna) in an apparently healthy person, which tends to recur, and is not accompanied by hæmatemesis.

2. Severe pain in the right hypochondrium, two or three hours after a meal.

3. Local tenderness.

4. Severe gastralgia.

PERFORATION is indicated by the *sudden* occurrence after a meal, or severe exertion, or during vomiting, of intense pain in the upper part of the abdomen, with rigidity of its walls, faintness, rapid wiry pulse, pinched and anxious face. The abdomen is much distended and the hepatic and splenic dulness are often absent. Later there may be fluid in the flanks.

SUBPHRENIC ABSCESS forms between the diaphragm and the liver, stomach, and spleen. After the initial symptoms of perforation, the physical signs are displacement of the cardiac apex, slight bulging of the affected side, cessation of abdominal breathing, and displacement downwards of hepatic dulness. Pulmonary complications are common.

Treatment.—What are our indications in such a case? Obviously (1) Improve the general hygienic surroundings; (2) Give the stomach rest; (3) Promote the healing of the ulcer; (4) Improve the blood condition; (5) After the acute symptoms are over, improve the local supply of blood by gentle massage to the stomach.

Diet.—A wine-glassful of milk, with a little lime-water added, every two hours, must be at first the only food allowed by mouth. It may, however, be supplemented in some cases by nutrient enemata of pancreatised beef extract. Where there has been recent hæmatemesis, or where there is severe pain, feed for some time entirely by the rectum. Strict rest in bed must be enjoined.

Medicinal.—The favourite remedies are bismuth (powder), with morphia, dilute hydrocyanic acid, and the administration of Carlsbad salts. The former act mechanically by coating the ulcer, and also have an anodyne effect upon the pain, the last, by depleting the portal circulation, removing the constipation, and adding to the alkalinity of the blood. Hyperacidity may be met by giving bicarbonate of soda with the bismuth.

Note.—When the healing process has fairly advanced, the patient may have bland broths, raw meat sandwiches, whipped cream and eggs, etc. Pepsin is often of great use. The great drawback to successful treatment is the lack of co-operation on the part of the patient as regards diet.

Surgical measures are called for in the event of perforation, subphrenic abscess, repeated or very severe hæmorrhage, and in cases attended with pyloric stenosis (gastro-enterostomy).

CANCER OF THE STOMACH.

Cancer of the stomach is by no means a rare condition, and most varieties have been found at one time or other; the principal primary forms however are—

1. Malignant adenoma at the pyloric and the cardiac end.
2. Scirrhus cancer; this form constitutes the bulk of the pyloric growths.
3. Encephaloid.
4. Colloid.

3 and 4 are in all probability degenerations of the other two. Epithelioma at the cardiac orifice is probably an œsophageal cancer which has extended.

Ætiology.—The majority of cases occur between the ages of forty and sixty years. The disease is somewhat more frequent in males than females. It is said that in about 5 per cent of cases of simple ulcer, cancer develops subsequently, but there is no definite relationship to other diseases of the stomach.

Pathology.—As the pylorus is the most common site, and hard scirrhus the most common variety of cancer in that region, we shall discuss its growth briefly.

The cancer usually begins as an overgrowth of the epithelial cells lining the gastric glands; the new growths infiltrate the submucous tissue, proliferate rapidly, and finally all coats are invaded, resulting in the formation of hard nodular masses. The pyloric opening becomes much narrowed, and the resulting stenosis gives rise to the marked dilatation of the stomach, so characteristic of pyloric cancer. The subsequent pathological course of the cancer does not differ from that of cancer elsewhere. It may remain more or less stationary for a time, then undergo one of the various degenerations that morbid growths are liable to—*i.e.*, colloid degeneration, ulceration, etc. If we get a clear idea of these changes, remember the anatomical site,

and the physiological importance of the neighbouring viscera, there cannot be any doubt as to what symptom may or should crop up. The ulcerative process may invade important blood-vessels, cause hæmatemesis, or form adhesions with other organs, and seriously handicap their functions by actual invasion (see Cancer of the Liver), and lastly aggravate or hasten the malignant cachexia.

Symptoms.—They are for a time most insidious, but sooner or later the persistent vomiting, hæmatemesis, constant pain, emaciation, and cachexia, with the presence of a local tumour, declare the true condition. The symptoms are considered in detail in the diagnostic table appended.

Free HCl is present in the gastric contents in diminished amount, or may be altogether absent in the later stages. This is by no means characteristic of cancer, as a similar condition may exist in gastric catarrh, for example; but in any case where the symptoms suggest cancer, if free HCl is found to be present in normal quantity, this speaks very strongly for the non-malignancy of the affection.

Prognosis.—The disease is usually fatal in from six months to two years, but severe complications may bring about a fatal issue very speedily.

Physical Examination.—A tumour may be discovered three or four months after the onset of symptoms. It is found anywhere within an area formed by joining the ensiform cartilage with the umbilicus, and the umbilicus with the ninth costal cartilage. The tumour is nodular and painful on handling; it may receive an impulse from the abdominal aorta, and though at first it is somewhat mobile, it afterwards becomes fixed.

It must be noted that in a considerable number of cases there may be no tumour present from first to last.

Physical examination also reveals the dilated condition of the stomach. It should, however, be remembered, when the body of the stomach is involved, that the stomach may be atrophied and hour-glass-shaped instead of being dilated.

Diagnosis.—

CANCER.

1. Is rare under forty years of age.
2. Epigastric pain is more or less continuous, and not much relieved by vomiting.
3. Anorexia is marked.
4. Vomiting is not frequent, but is copious; and in the vomited matter may be found—*Sarcinæ ventriculi*, torula, etc. Cancerous fragments (rare). Free HCl is diminished or absent.
5. Hæmorrhage is seldom copious, but may be frequent (“coffee-ground” vomiting); most common in the later stages.
6. Loss of flesh; the development of cachexia is rapid.
7. Epigastric tumour is usually easily detected.

ULCER OF STOMACH.

1. Usually in young adults, especially women.
2. The pain is rendered worse by food, and vomiting gives much relief. The pain is also more localised and xiphi-vertebral in character.
3. Appetite is good, but the patient is afraid to eat.
4. Vomiting is frequent, and the vomited matter contains free HCl often in excess.
5. Hæmorrhage is not so frequent, but is copious at times. May be the first symptom.
6. When the gastric symptoms are severe and prolonged, a cachectic appearance may develop, but never to the same extent as cancer.
7. No epigastric tumour, though there may be some thickening of the walls.

Treatment.—Must be mainly palliative. Easily digestible food and hypodermic injections of morphia help to make the patient's life tolerable. Bacterial fermentation should be prevented by washing out the stomach once daily with a solution of boric acid (3iv to Oi), and the administration of anti-fermentatives, such as hyposulphite of soda, creosote, or glycerine of carbolic acid. Surgical procedures seem to offer some chance of prolonging life. Dr. Mayo Robson says:—When the disease involves the pylorus there are three courses open—

1. Complete removal by pylorotomy with suture of the ends of the stomach and duodenum.

2. Pylorectomy with closure of the open ends of the duodenum and stomach by invagination and suture, the communication between the stomach and bowel being that effected by gastro-enterostomy.

3. In extensive disease, or where the patient is unable or unfit to bear the major operation, gastro-enterostomy may be performed, the stomach and jejunum being fixed in apposition by means of Senn's plates.

HÆMATEMESIS

Vomiting of blood may be an important symptom in many diseases.

The source of the hæmorrhage may be either congested and distended venules or capillaries in the gastric mucous membrane, or the bursting of a large vessel; for example, the splenic artery in gastric ulcer.

Causes.—The more common are—

1. Erosion of vessels by gastric ulcer, cancer, chronic gastritis.

2. Congestion of the portal circulation from any cause, but especially cirrhosis of the liver, extreme backward pressure from cardiac disease, cancer and other growths in the liver.

3. The action of irritant poisons.

4. Alterations in the blood and blood-vessels, by which blood oozes through, as seen in malignant fevers, purpura, severe jaundice, etc.

5. Blood that has been previously swallowed may be afterwards vomited (epistaxis, pulmonary hæmorrhage, etc.).

Symptoms of course depend on the extent of the hæmorrhage; when it is severe the symptoms of collapse are marked. The attack is usually sudden, and accompanied by a feeling of intense nausea and a feeling of weight in the stomach. In ulcer it frequently follows a meal or sudden exertion. After the collapse has passed off a stage of reaction occurs.

Diagnosis.—When the hæmorrhage is not profuse it is sometimes difficult to distinguish this condition from bleeding from the lungs.

I append a table showing the chief differences:—

HÆMOPTYSIS.	HÆMATEMESIS.
1. Previous history of pulmonary troubles.	1. Previous history of gastric disturbance.
2. Blood is coughed up.	2. Blood vomited.
3. Blood is frothy and bright red.	3. Blood is dark coloured and not frothy.
4. Blood may be mixed with sputa.	4. Blood may be mixed with food.
5. Dyspnœa and pains in the chest.	5. Nausea, and weight in epigastrium.
6. Is not usually succeeded by melæna.	6. Often followed by melæna.

Whilst the above differences are of the utmost value, they are not always conclusive. For instance, in cases of hæmoptysis the blood is frequently *not* frothy, or an amount of the pulmonary blood may be swallowed and *vomited afterwards*. Again, the hæmorrhage may be so severe, so sudden, and so inexplicable as regards its real cause, that a hasty judgment may be quite erroneous.

Treatment.—The first thing to be decided if possible is the source of the bleeding; and secondly, whether it is advisable to check the hæmorrhage, knowing that hæmatemesis is so often a safety-valve in portal congestion. Having decided to check the hæmorrhage, absolute quiet, mentally and bodily, must be obtained, and no treatment is more efficacious than a full hypodermic injection of morphia, combined with the administration of ice and dilute sulphuric acid. Feed by the rectum for the first few days, and afterwards give all food *cold*. If reaction does not follow, and the state of collapse persists, subcutaneous transfusion of warm normal saline solution may be performed. One to two pints may be injected. In very severe and repeated hæmorrhages operation with ligature of the bleeding vessel may save life.

DILATATION OF THE STOMACH.

This condition may be obstructive or non-obstructive. The latter form is due to defective innervation or constant distension by excess of food, giving rise to atony of the muscular coat. Anæmia and acute febrile diseases therefore predispose to it. It is met with as a sequel of gastric irritation or insufficiency, and in chronic catarrh.

Obstructive dilatation is caused by (1) stenosis of the pylorus (cancer, cicatrising ulcer), (2) pressure on the duodenum or contraction after duodenal ulcer, (3) constriction of the pylorus in chronic peritonitis, (4) constriction of the cardiac end by adhesions.

In great dilatation there is bacterial fermentation, owing to delay of food in the stomach. In the lesser degrees there may be hyperchlorhydria (gastric irritation) or hypochlorhydria (gastric insufficiency and catarrh). If the obstruction lie beyond the pylorus there is hyperchlorhydria, and bile is present in the vomit.

Symptoms occur at irregular intervals; it may be several hours after food, or at the end of the day, or several days may elapse between attacks. There is diffuse burning epigastric pain (*pyrosis*), relieved by vomiting, which patients often excite. Flatulence and constipation are marked. Wasting is present. Both respiration and circulation are affected. Where dilatation is marked, the outline of the greater curvature, and sometimes of the lesser also, may be visible. By forcibly stroking the epigastrium, peristaltic movements of the stomach may be set up. On shaking the patient's body from side to side, a splashing sound (*succussion*) can often be heard, due to the mixture of gas and fluid in the stomach. It is not of diagnostic value unless heard after the time at which food should have normally passed into the intestine (5-6 hours after the last food or drink). To determine the size of the stomach it must be artificially distended by fluid or by gas. Air may be pumped in, or 30 grs. of tartaric acid may be given in half a tumbler of water, followed by 30 grs. of

bicarbonate of soda in the same quantity of water. CO_2 is thus developed in the stomach. The greater curvature can then be percussed out, giving a dull note if fluid has been used, if gas a tympanitic note. Auscultatory percussion may also be employed.

The vomited matters are characteristic. The quantity is larger than the normal content of the stomach, and the taste is excessively sour (in the early stages sometimes from hyperchlorhydria, later from defect of HCl and excess of organic acids). Fragments of partly digested food are found, and under the microscope the bacillus acidi lactici, bacillus butyricus, and sarcina ventriculi.

Prognosis is that of the causative disease, though appropriate treatment always relieves symptoms.

Treatment.—Careful regulation of the diet, avoiding carbohydrates where there is much bacterial fermentation. In non-obstructive cases daily lavage of the stomach with anti-fermentative or alkaline solutions (boric acid, permanganate of potash, bicarbonate of soda) continued till the returning fluid is clear, then administration of antifermentatives (Sodii Hyposulph., Glyc. Acid. Carbol., Creosote, etc.). Massage and douching of the abdomen.

In obstructive cases washing out is only palliative. Gastro-enterostomy or pylorotomy should be done as soon as the diagnosis is made.

ATROPHY OF THE STOMACH

May occur in long-continued exhausting diseases, cancer of the cardiac end of the stomach, bulbar paralysis, or malignant stricture in any part of the œsophagus. The symptoms are largely masked by those of the primary disease, and the diagnosis is rarely made during life.

DYSPEPSIA

Is a functional disorder of the stomach resulting in disordered activity, and causing faulty digestion of food. Its

main types are three:—(1) cases where the direct irritation of the food plays a prominent part (*gastric irritation*), (2) cases where there is a primary functional defect in the stomach dependent upon such general diseases as anæmia, etc. (*gastric insufficiency*), (3) cases where the symptoms are mainly dependent upon disorder of the nervous system (*nervous dyspepsia* or *gastric neurosis*). The above is Sidney Martin's classification, but there are many others, as acid, atonic, and flatulent dyspepsia, and so on. All three classes tend to run into one another, and although strictly functional at first, may end in the organic changes of chronic gastric catarrh.

The following Table may assist the reader in diagnosing the two conditions:—

	CHRONIC GASTRITIS.	DYSPEPSIA.
<i>Pain</i>	Often severe with diffuse epigastric tenderness.	Less severe; tenderness is usually absent.
<i>Fever</i>	Temperature sometimes slightly raised.	Not raised.
<i>Thirst</i>	Often a marked symptom.	Absent. •
<i>Vomiting</i>	Frequently, especially in the morning. Lactic, butyric, and acetic acids often present. Pain is not usually relieved by vomiting.	Vomiting is not frequent except after certain foods, then relief is obtained.
<i>Causes</i>	Usually the constant introduction of irritants, such as alcohol in excess, abuse of tea, morphia, etc.	See list of causes tabulated. Often there is no obvious cause and the best dietetic treatment may fail to cure.
<i>Tongue, etc.</i>	Is furred, red at the tip and edges. The lips are cracked, and the gums spongy and red.	Tongue broad, flabby, and indented by the teeth. Gums are soft and anæmic. Lips are not usually fissured.
<i>Morbid Anatomy</i> . .	Stomach is much thickened, the mucous membrane is often much atrophied, and fibrous in structure. It presents a rough mammillated appearance with suppurating points, localised vascular areas, and hæmorrhagic erosions. <i>Note.</i> —Though the membrane is thickened, there is marked atrophy of the glandular elements.	In pure dyspepsia these changes are not present. The mucous membrane may be thickened and injected. The muscular fibres are pale, flabby, and relaxed.

Causes of Dyspepsia.

Faults on the part of the organs—

1. Bad teeth, causing defective mastication.
2. General debility of the digestive organs after fevers, etc.
3. Deficiency in the quantity or quality of the gastric juice, pancreatic secretion, and bile.

Faults on the part of the patient—

1. Habitual use of the *same kind* of food.
2. Intemperance in eating and drinking.
3. Excessive use of tobacco, especially if attended with excessive expectoration of saliva.
4. Bolting food.
5. *Cold* drinks during meals.
6. Mental work immediately after eating.
7. "Dirty" teeth.
8. *Deficiency of food*.
9. Excessive use of tea, coffee, alcohol, etc.
10. *Sound* sleep after dinner.

By the above table it will at once be seen that it is most difficult to enumerate all the causes of indigestion; but it should be remembered that many persons may eat with impunity what would cause much distress in others.

The more common symptoms are pyrosis, flatulence, eructations of acrid matters, disagreeable breath, vertigo, etc.

Many forms of dyspepsia are described, and I append a table showing the principal points of the three more distinct forms; at the same time it must be remembered the table is only a guide, for the types usually overlap each other.

	ATONIC DYSPEPSIA. (Gastric Insufficiency.)	ACID DYSPEPSIA. (Gastric Irritation.)	NERVOUS DYSPEPSIA.
<i>Immediate Cause.</i> . .	Want of functional power, both as regards gastric secretion and movements. Hence often secondary to constitutional diseases.	Usually primary, but may follow other diseases. Dependent on errors of diet, drink, etc.	Mental strain from worry, overstudy, hysteria, neurasthenia, etc.
<i>Pain, vomiting, etc.</i>	Fulness and oppression in chest after meals; vomiting absent.	Dull pain some time after food; nausea and vomiting.	Often severe gastralgia, <i>relieved</i> by food; but may simulate pain of ulcer. Vomiting not common.
<i>Eructations</i> . . .	Eructations not frequent, but flatulence very marked. Often some dilatation of stomach.	Flatulence common.	Eructations of gas or fluid very marked, and flatulence extreme. Hiccough very frequent.
<i>Examination of gastric contents</i>	Deficiency of HCl. Excess of lactic acid.	Excess of HCl, and sometimes of lactic and butyric acids.	Secretion of HCl variable; often in excess, at other times deficient.
<i>Tongue</i>	Broad, flabby, papillæ raised, furred at the back, and tremulous.	Broad also, but usually coated with a thick yellowish fur. Saliva increased at first, mouth afterwards dry.	Is usually clean, raw - beef - like in character, pointed tip, firm, not flabby.
<i>Urine</i>	Normal or high coloured from urates.	High-coloured deposits, "gravel," and oxalates.	Pale deposit of amorphous phosphates.
<i>Special points.</i> . .	Most common amongst young women. Apt to persist.	Most common amongst middle-aged people of generous build. Paroxysmal in character, migraine and mental depression marked during the attack.	Most common in neurasthenics, or those subject to nervous alterations. Little influenced by treatment, the predisposition remaining. Insomnia a prominent symptom, and other nervous disturbances common.

Treatment.—Stock prescriptions and rigid diet charts account for many of the failures in treating dyspepsia. Dyspepsia is not a disease, it is symptomatic of an altered digestive tract, and it is our first duty to find out the most probable cause, then, having fully considered the constitution

of the patient, to advise careful dieting, fresh air, exercise, etc. It is at all times our duty to suggest the avoidance of those things we know are indigestible, but he is a conceited man who thinks he knows better than the patient what best agrees with him. What ought to agree is often the thing that does *not*. If the cause be obscure, and the treatment slow in producing benefit, a careful examination of the gastric secretion after a standard meal should be made. The main points to remember are—

1. First allay any undue irritability of the stomach by a restricted amount of food, and the administration of hydrocyanic acid, bismuth, and morphine in an effervescing form.

2. In gastric irritation, where there is much pain or eructation, give alkalies after meals; in gastric insufficiency, promote gastric secretion by giving alkalies before meals; or imitate the secretion by giving pepsin and hydrochloric acid after meals.

3. Promote healthy peristalsis by giving muscular tonics such as nitro-hydrochloric acid with liquor strychninæ.

4. Keep the bowels open.

5. Avoid turning the patient's stomach into a chemist's shop.

6. If there be much fermentation and evidence of the collection of ropy mucus in the stomach, wash it out and give a pill containing a minim or two of carbolic acid twice a day for a short period.

Remember that a healthy co-operation on the part of the patient is necessary, and this can only be obtained by showing him that we thoroughly understand what is wrong. When the patient is highly neurotic, a blister over the epigastrium is of great value.

IV. THE INTESTINES.

ENTERITIS.

It is somewhat difficult to define what is meant by enteritis, for the mucous membrane of the intestines is inflamed in very many diseases, especially in cholera, typhoid fever, dysentery, etc. But for clinical purposes, the term is much more strictly

limited, and by enteritis is meant an inflammation of the alimentary tract, due to irritation by indigestible food, or a general catarrhal condition following exposure to cold, etc.

As in all other inflammations of mucous membranes, we may get the following varieties:—

1. The ordinary, or catarrhal inflammation.
2. The croupous or fibrinous form, when the inflammatory exudation is unusually rich in fibrin, and tends to coagulate.
3. The phlegmonous type; when the submucous coat is deeply involved, and presents a number of suppurative points which burst and form ulcers.

It should be noted that in some forms of enteritis the actual inflammatory changes are very slight, and the symptoms are mainly due to bacterial intoxication.

INTESTINAL CATARRH.

Acute Catarrh is most frequent in the hot summer months, possibly because of the ingestion of large quantities of unripe or decomposed fruit, sour milk, etc. It is evident that by excessive decomposition in the intestines there is formed a favourable nidus for the multiplication of bacteria, which otherwise would lie dormant. Other causes are prolonged administration of mercury or arsenic, too vigorous purgation, and excess in strong alcoholic drinks.

Pathology.—The more marked changes are observable in the *ileum* as hyperæmia of the *valvulæ conniventes*, a swollen condition of the solitary glands which project like small shot, and sometimes present abraded or slightly ulcerated surfaces. The mucous secretion is much increased, and a slight amount of pus may be formed.

Chronic Catarrh most frequently is due to alcoholism, portal congestion, atony of the gut from constitutional diseases, habitual constipation, etc.

Pathology.—The more marked changes are observable in the *large gut*. The mucous membrane is much thickened, often pigmented, and shows extensive small follicular ulcers, which

after healing give a peculiar worm-eaten character to the gut on holding it up to the light. The superficial veins are distended, piles are often prominent in the rectum, and polypoid outgrowths may spring from the mucous surface.

Symptoms of ACUTE Enteritis. The chief symptoms are—

1. Diarrhœa.
2. Abdominal pain, especially around umbilicus.
3. Nausea, anorexia, and vomiting, *without fæcal odour*.
4. Pyrexia.

The above are the so-called cardinal symptoms, but the inflammatory condition, if severe, may be complicated with peritonitis, owing to the permeation of the intestinal wall by bacteria, or more frequently by their toxins or by ptomaines. Taking a typical case of enteritis, the diarrhœa is the chief symptom, and as in such cases excessive decomposition takes place in the intestines, we naturally expect the stools to be offensive, and to consist principally of undigested food, epithelial debris, triple phosphates, biliary pigments, and mucus. Various organisms, such as the *proteus vulgaris*, the *bacillus enteritidis*, and the *bacillus coli*, may also be found. A word of caution is necessary, as regards bile pigments. The fæces may appear to contain an excessive amount of bile through the haste with which the duodenal contents are hurried on; or on the other hand, the same catarrh that caused the enteritis may block up the common bile-duct and prevent the bile from entering the bowels, in which case the fæces will be pale and clay-like, and a subsequent jaundice may develop. Without a careful examination of the stools, it is impossible to diagnose whether the morbid process is confined to the duodenum, jejunum, or ileum.

In children the fæces may have a grass-green colour. The temperature in them rises sharply to 103° or even 105°.

The general symptoms are marked. The face is pinched, the tongue dry and furred. The pulse is disproportionately rapid. Thirst and oliguria are present. In fatal cases

collapse ending in coma appears, often without warning. It is due to toxæmia.

Treatment.—First clear out any offending matter with castor oil or a mercurial purge. Relieve pain with an hypodermic injection of morphia, and the application of hot poultices or turpentine stupes to the abdomen. Douche out the colon with warm saline solution passed slowly into the rectum. Obviate collapse by rectal stimulation or strychnine hypodermically. An effervescing mixture containing hydrocyanic acid and bismuth is useful in allaying undue irritability of the stomach. Salol or β -naphthol are our best intestinal disinfectants.

Symptoms of CHRONIC Enteritis.—

1. Hæmorrhoids.
2. Constipation, alternating with diarrhœa.
3. Discharges of blood and mucus from the anus.

The fæces do not present the same “undigested food” character as in acute enteritis, but there is more tenesmus or violent straining at stool. The fæces often resemble the chronic dysenteric stools—viz., boiled sago streaked with blood; but, unlike dysentery, the diarrhœa is easily provoked by hot liquids, and only too often by *any* meal; this shows that the irritable state of the gut is by no means localised to the large gut, though the more marked pathological changes occur in that part of the alimentary canal.

Treatment.—Remove any obvious cause. Salol in 15-grain doses nightly with the addition of 3 minims of liq. arsenicalis and 10 minims of tinct. opii before each meal, occasionally acts like a charm. If these measures do not succeed, treat according to the principles laid down under Chronic Dysentery.

Croupous and phlegmonous enteritis occur chiefly in the final stages of chronic constitutional diseases (granular kidney, etc.), and are generally fatal. The treatment is purely symptomatic.

TYPHLITIS, PERITYPHLITIS, AND APPENDICITIS.

By typhlitis is meant inflammation of the cæcum proper; by perityphlitis is meant a phlegmonous inflammation in the cellular tissue uniting the cæcum with the psoas and iliacus muscles; and by appendicitis is meant inflammation of the appendix vermiformis.

It appears, however, that the former condition is merely an extension of appendicitis, and it is becoming more and more recognised that the term typhlitis ought to be abolished, the terms perityphlitis and appendicitis being sufficiently comprehensive. We shall first consider the anatomical peculiarities of this region.

1. The cæcum marks the junction of the small with the large intestine, the junction being guarded by the ileo-cæcal valve, which allows fæces, etc., to pass from the small to the larger gut, but at the same time prevents regurgitation. The ileum joins it on its inner aspect about two-and-a-half inches above its blind end.

2. The cæcum is not invested posteriorly by peritoneum, but by loose cellular tissue.

3. The appendix vermiformis lies in close proximity.

The vermiform appendix is situated at the inner, lower, and back part of the cæcum, and usually looks towards the spleen. Unfortunately for clinical purposes, the exact position can scarcely be diagnosed during life, and *post mortem* it has been found in almost every region of the abdomen.

Solid bodies of all kinds may find their way into the appendix. Osler asserts that "foreign bodies rarely lodge in it, but that concretions of inspissated mucus and fæces in which lime salts are deposited, forming enteroliths, are common."

APPENDICITIS

Usually occurs in young people, and oftenest in the male.

Causes.—Foreign bodies, concretions, tubercular ulceration, malignant growths, constipation, habitual use of indigestible food, etc. Even without injury due to such causes, virulent bacteria may of themselves cause inflammation.

Pathology.—

Varieties.—1. The catarrhal form.

2. The ulcerative variety (apt to end in perforation).

In the catarrhal appendicitis the tube is thickened as a whole, the muscular walls are somewhat thickened or fibrosed, and the mucous coat is covered with a thick viscid mucus. The serous coat may become adherent to the neighbouring peritoneum, forming adhesive bands, which may cause a loop of gut to be strangulated or to kink over them.

In chronic catarrh the lumen of the tube may be partially obliterated by adhesions, causing the formation of an appendicular cyst, which may ultimately rupture into the peritoneal cavity.

The ulcerative variety shows, in addition to the above changes, one or more ulcers; moreover, the ulcerative process is followed by most important results.

1. *Perforation may occur AFTER adhesions have formed*, and be followed by circumscribed intra-peritoneal abscess. This localised abscess may after a time open into the peritoneal cavity, and excite a most intense general peritonitis.

2. *When the appendix is NOT WITHIN the general peritoneum*, perforation produces a retro-peritoneal abscess; in such cases, the pus may burrow in the cellular tissue, between the gut and the iliac fascia, and may then extend in three directions—

- (1) Upwards, forming a perinephric abscess.
- (2) Downwards to Poupart's ligament, where it is prevented from extending down the thigh, by the union of the ligament and fascia, and so bursts externally.
- (3) Into the true pelvis, when it may burst into the rectum or bladder, or through the obturator foramen.

3. *Perforation may occur BEFORE adhesions are formed*, the appendix hanging free in the peritoneal cavity. In such cases, a speedy death results from the violent septic peritonitis.

Symptoms.—In the *catarrhal variety* the chief symptoms at first are sudden onset of localised pain in the right iliac fossa, local tenderness, greatest at M'Burney's point (one third of the distance between the right anterior superior iliac spine and the umbilicus), elevation of temperature, furred tongue, and vomiting, not excessive, and not faecal; later, if suppuration occurs, a tumour may be detected above Poupart's ligament, possibly occupying the whole of the right iliac fossa; it may be absolutely dull on percussion, or give a modified resonant note. The pain is often paroxysmal, and shoots down the right leg, which is flexed; the temperature becomes septic in character; constipation is usually a marked feature, but there may be diarrhoea, painful micturition, etc. In the ulcerative form, when perforation occurs into the peritoneum, the symptoms are those of shock, or those described under general peritonitis.

An ordinary attack of appendicitis passes off in from seven to ten days, the inflammatory thickening disappearing completely. Should pus form, however (perityphlitic abscess), the fever continues or increases, and the swelling becomes larger, harder, and more tender. Fluctuation is very rarely present.

Diagnosis.—The diagnosis is often very difficult, and the symptoms may simulate obstruction of the bowels very closely; but in appendicitis the tumour is diffuse, and faecal vomiting is rare, or, according to some authorities, never present. In children the history, tenesmus, bloody discharge, and rectal

examination under chloroform, make a diagnosis of *intussusception* fairly easy, though it sometimes simulates appendicitis. Psoas abscess, and pelvic cellulitis in women are also conditions which must be eliminated before arriving at a diagnosis.

Where the appendix extends downwards into the pelvis, the existence of tumour may be made out only on rectal or vaginal examination.

After an attack of appendicitis has passed off, there is great liability to recurrence upon slight provocation, and such recurrences may be many times repeated.

Treatment.—*During the acute attack*, if fever is slight and there is no great local swelling, it may be unnecessary to operate. In such cases hot fomentations, enemata to open the bowels, morphine if need be for the relief of pain, fluid diet (chiefly milk), and absolute rest in bed are the chief means of treatment. In convalescence the general health must be as far as possible built up to meet the ensuing operation. It is now the practice to *remove the appendix* after a first attack, even where there is complete recovery, for the liability to recurrence is great, and any recurrence may lead to perforation. In cases with severer symptoms, and even in those that are doubtful, operation is done during the first attack. It is of course necessary where perityphlitic abscess exists, and where there is generalised peritonitis. In the last case it affords the only hope of recovery, though the hope is but slender.

INTESTINAL OBSTRUCTION

May be due to many causes, as the following Table taken from Dr. Tanner's work shows.

1. *Inter-mural*, or those originating in, and implicating, the mucous and muscular coats of the intestinal walls—

- (1) Cancerous stricture.
- (2) Non-cancerous stricture, comprising—(a) Contraction of cicatrices, following ulcerations. (b) Contraction of walls of intestine from inflammation, non-cancerous deposit and injury.

(3) Intussusception.

(4) Intussusception, associated with polypi.

2. *Extra-mural*, or those causes acting from without, or affecting the serous covering—

(1) Bands and adhesions from effusion or lymph.

(2) Twists or displacements.

(3) Diverticula.

(4) External tumours or abscesses.

(5) Mesocolic and mesenteric hernia.

(6) Diaphragmatic hernia.

(7) Omental hernia.

(8) Obturator hernia.

3. *Intra-mural*, or obstructions produced by the lodgment of foreign substances—

Foreign bodies, hardened fæces, concretions having for their nuclei gall-stones, etc.

As the subject of intestinal obstruction really belongs to the domain of surgery, only the main clinical points will be condensed here.

The obstruction may be of an acute or chronic nature.

General Symptoms of any Complete Obstruction.—The more common are pain, distension of the abdomen, and stercoraceous or faecal vomiting, with complete constipation.

The pain is variable, but is usually intense, at first paroxysmal, but later, continuous. It is not always referred to the part obstructed.

The constipation is absolute, but the bowel *below* the stricture may pass fæces, or the masses may be removed by an enema.

The vomited matter consists at first of the stomach contents, then bilious matter, and finally faecal matter, which may in addition be tinged with blood.

An important symptom is paroxysmal peristaltic movement of the coils of intestine above the obstruction. This is visible through the abdominal walls.

The patient, if unrelieved, falls into a typhoid state from intense peritonitis, etc., and usually succumbs in from four to six days after the onset of the severe acute symptoms.

In *chronic obstruction* (most commonly due to malignant tumour) there is a history of gradually increasing constipation, alternating, it may be, with attacks of diarrhoea. The fæces are narrowed in calibre. They may assume a pipe-stem shape or be flattened like a tape-worm, or they may be passed in small rounded masses like sheep's dung. Frequently they are smeared on the surface with blood and pus. Portions of the growth are sometimes found.

Special Forms of Obstruction.—

I. INTUSSUSCEPTION

Occurs most frequently at the ileo-cæcal valve. Usually the ileum and cæcum, preceded by the valve, pass into the larger colon. The valve forms the apex of the intussuscepted gut, and is an important aid to diagnosis on rectal examination.

Causes.—Severe and sudden peristalsis, especially in young children.

Special Symptoms.—A sausage-like tumour felt through the abdomen, glairy mucoid and bloody discharge, and tenesmus. The tumour frequently travels from right to left, describing a semicircular curve round about and above the umbilicus. It can sometimes be felt in the rectum. Spontaneous reduction may take place, or the gangrenous invaginated portion may slough and pass per anum.

Treatment.—Gentle manipulation under chloroform, or copious injection of hot water into the rectum may sometimes cause the intussuscepted portion of bowel to recede. In the vast majority of cases laparotomy is necessary, the earlier the better. Other means of treatment should only be tried when surgical aid cannot be got at once. Prolonged manipulation lessens the patient's chances.

II. VOLVULUS

Is a twisting or bending of a coil of intestine in such a manner that its calibre is obliterated at that spot. Is most common in the sigmoid flexure, and towards the back of the abdominal cavity.

Special Symptoms.—Volvulus furnishes the most typical symptoms of acute obstruction.

Treatment.—Operative.

III. IMPACTION OF FÆCES, AND OTHER FOREIGN BODIES.

Treatment.—Try clearing out the lower bowel with copious enemata, breaking up hardened fæces, massage under chloroform, etc., prior to laparotomy.

It will be seen from the foregoing statements that as the relief of intestinal obstruction usually resolves into surgical measures, the reader should look for details of these important conditions in a work on surgery. As, however, these cases frequently first come under the notice of the physician, and also as the patient and his or her friends frequently object to operative measures, the physician must take a determined stand or attitude; for he must remember every hour of delay in obtaining relief is fraught with extreme danger, and he should therefore lose no time in advising laparotomy the moment he is convinced that the obstruction is complete. We may, however, temporise in cases of fæcal accumulation, and in the more chronic forms of intestinal obstruction met with in old people, in malignant disease, etc., where surgical measures are little less than hopeless.

When we decide to temporise, we must put the patient under the most favourable circumstances we can—alleviate the pain by opium, the distressing vomiting by ice, and apply hot fomentations to the tender abdomen. Laxatives or purgatives should never be given. Enemata, administered very slowly, are harmless, and are often of assistance in deciding whether an obstruction is actually present.

Where operation is decided upon, no time should be lost after the diagnosis is made. Even in the most favourable circumstances the mortality is very great. No cases require graver thought than intestinal obstruction, and if there be the slightest chance of only temporary relief by surgical procedure, the patient should have his choice.

TUMOURS OF THE INTESTINES.

Simple tumours are very rare. Sarcoma and lymphosarcoma occur, but by far the most frequent tumour is

CANCER. This affects the large intestine principally, especially the various flexures of the colon *and the rectum*. It is commonest in the male, between 40 and 60, but it may arise as early as 20 years of age.

Morbid Anatomy.—Usually a cylinder-celled carcinoma. The growth is annular, and tends to early ulceration. Metastases may be found in the mesenteric and other intra-abdominal glands, or a generalised peritoneal carcinosis may occur. It may spread by the blood-vessels through the portal vein to the *liver*.

Symptoms.—

1. The general symptoms of cancer.
2. Sometimes ascites, or enlarged inguinal glands.
3. Pain of two kinds—a fixed dull pain more or less corresponding to the site of the lesion; and paroxysmal colicky pain, worst just before defæcation.
4. Obstinate constipation, or constipation alternating with diarrhœa.
5. In the late stages, reflex vomiting. Stercoraceous vomiting is uncommon.

Physical Signs.—

1. Tumour (present in 40 per cent of the cases). Hard, nodular, tender, *freely mobile*.
2. Meteorism, very common.

3. Visible peristalsis in the later stages, where obstruction is becoming complete.
4. Alterations in the fæces: diminution of their calibre if the stricture be low down; admixture of blood and pus, in large or only microscopical amount.

RECTAL CANCER.—Along with all the above symptoms, *pressure symptoms* may occur. The first evidence is usually interference with defæcation. The stools are altered in calibre (“pipe-stem” or “sheep’s-dung” stools). This is followed by tenesmus, and repeated unsatisfactory efforts at evacuation. Later mucus, blood, and pus become persistent in the stools.

Physical Signs.—Digital examination reveals the presence of a hard, irregular, nodular tumour.

Complications.—Chiefly *rupture* into neighbouring viscera, and metastases in *liver*.

Diagnosis.—One must not be satisfied with a mere diagnosis of rectal cancer, but must endeavour to determine the possibility of relief by operation. For this the extent of the tumour, the degree of mobility, and the presence of complications must be considered.

Differentially, syphilitic stricture is the main source of error. Here, however, the ulcers do not present the protuberant edges of carcinoma. They are *multiple*, and the process is usually diffuse. This form of stricture is more frequent in women, cancer in men.

Treatment of **INTESTINAL CANCER.**—No *cure* results from treatment, even in operable cases, for there is a liability to recurrence at any point of the after history. In inoperable cases treatment must be directed to (1) temporary increase of strength—tonics, and especially dietetic treatment; (2) removal of symptoms of stenosis by mild laxatives or appropriate changes in diet; (3) for the relief of pain, narcotics; (4) operative measures of a palliative nature. These are very various, according to the situation of the tumour, but rarely lead to more than temporary improvement. It depends upon the

individual temperament whether the last state of a man, whose symptoms of obstruction have been temporarily relieved by colotomy, may not be worse than the first. To any one at all sensitive, death, not long postponed in any case, is probably better than the constant dribbling of fæces over the back.

DIARRHŒA.

We have already seen that the frequent passage of loose motions from the bowels occurs in many diseases, but whilst diarrhœa is often associated with grave lesions of the intestines, such as cancer, typhoid, tubercular ulcers, enteritis, etc., it may come on without any apparent cause, or after slight nervous disturbance, fright, etc.; in other words diarrhœa may be merely a functional disorder, or symptomatic of a grave disease.

Causes.—Increased discharge of fæces can be induced by—

1. Increasing the intestinal secretions.
2. Exciting peristalsis, directly through irritants, or indirectly through the nervous system.

The more common causes independent of actual disease of the intestines are—eating indigestible food, unripe fruit, etc.; drinking cold liquids; certain nervous states, especially at the climacteric period. I once had a patient who had an attack of diarrhœa every Friday night after making his weekly visit to his bakehouses; the change from cold to heat was evidently the cause in this case.

Diagnosis.—It is not always easy to say whether the diarrhœa is a mere temporary disturbance, or the evidence of a disease, acute or otherwise. In all cases note—

1. How the looseness commenced.
2. The consistency, colour, and odour of the fæces. Look for blood, fat, calculi, etc.
3. Pain and abdominal tenderness.
4. The amount of pain at the time and immediately after defæcation.

Treatment.—Having satisfied ourselves that the diarrhœa is due to an irritant or irritating products still in the gut, clear out the bowels with a full dose of castor-oil, then we may give opium in various forms, a full dose of the tincture, Dover's powder, and bismuth, chalk mixture, etc.

Broadly speaking, *acute* diarrhœa is best treated with alkalies and opium; *chronic* diarrhœa with astringent minerals, or acids, with opium. Hot fomentations to the abdomen, diffusible stimulants if the patient feels chilly, and a restricted diet, are the main points to be remembered.

In that form of diarrhœa which comes on immediately *after* food in nervous people, nothing succeeds better than two minims of liq. arsenicalis with five minims of laudanum, given just *before* meals.

Diarrhœa in young children or infants, if not associated with other diseases, such as rickets, tubercular conditions, etc., is nearly always due to improper feeding, or irritation from worms.

CONSTIPATION.

To ensure the due evacuation of the bowels, the digestive functions of the stomach and small intestine, and the secretion of bile and pancreatic juice, must all be in proper working order; and further, the colon must absorb some of the water from the fluid fæces, rendering them of a proper consistency for expulsion. Evidently, then, constipation may be brought about by interference with any of these functions, and hence may be due to a large variety of causes. The chief are—

1. General diseases: anæmia, fever, chronic Bright's disease, etc.
2. Nervous diseases: paraplegia, chronic brain disease.
3. Sedentary habits.
4. Causes inducing loss of water (diabetes, perspiration).
5. Change of diet or habits.
6. Painful defæcation.
7. Obstructive jaundice.

Anatomy.—The changes in the gut are few. There may be hypertrophy of the muscular coat of the descending colon, and small ulcers in the cæcum. In extreme cases there may be enormous distension of the whole colon.

Symptoms.—Sometimes practically absent. Usually headache, anorexia, depression, and loss of energy. Where the lower bowel is much loaded there may be *pressure* on the lumbar or sacral nerves, and pain down the back or front of the left thigh. Pressure on the intrapelvic veins may cause hæmorrhoids or varicocele.

Fæcal masses may sometimes be felt by palpation through the abdominal wall, or rectal examination may reveal the presence of hard scybala.

Treatment.—A daily attempt at evacuation should be made at a fixed hour. Regular exercise must be taken. Correct any constitutional fault. Add laxative vegetable foods to the diet. Give a tumblerful of water before breakfast, and an occasional laxative. Even in severer cases *avoid purgation*, but use a mild laxative habitually. The following is an excellent pill:—

R	Aloin	gr. $\frac{1}{4}$.
	Strychninæ	gr. $\frac{1}{60}$.
	Ext. Belladonnæ	gr. $\frac{1}{16}$.
	Ext. Cascaræ	gr. ss.

Sig.—One pill thrice daily.

or give aperient waters in the morning.

If the rectum is blocked by scybala these must be removed mechanically, and soap and water enemata given daily for some time afterwards.

INTERNAL PARASITES AND PARASITIC DISEASES.¹

Definition.—Parasites are low forms of organisms—animals or vegetables—which infect other animals or plants, lodging

¹ I am much indebted to my friend Dr. RYLAND WHITAKER for valuable help in this portion on Parasites and Parasitic Diseases.

upon them or within them, and deriving their nourishment from the tissues and juices of their host.

Division.—Parasites are divided into two great classes—

1. Animal Parasites.
2. Vegetable Parasites.

ANIMAL PARASITES.

General Characters.—Animal parasites belong to the lowest types of the animal kingdom, and are characterised by the greater simplicity of their structure as compared with that of the allied nonparasitic forms.

Life History.—

1. *Metamorphosis.*—Most animal parasites exist in two or more forms: the immature, larval, or embryonic form, and the mature form. Many of them undergo a series of changes—alternation of generation—before reaching maturity; first, by sexual union producing ova which give rise to embryos or larvæ, which ultimately develop into adult forms, or by a process of budding, produce colonies of the mature organisms.

2. *Host.*—The animal infected by the parasite is called the host.

3. *Intermediate Host* is the organism in which the immature forms are lodged.

4. *Habitat* is the part of the body of the host in which the parasite, or its immature form, takes up its abode.

The animal parasites of the human body, exclusive of microscopic organisms, belong to the order *Vermes*. They are of three classes, *Cestoda* (tapeworms), *Nematoda* (round worms), and *Trematoda* (flukes).

CESTODA.

(Κεστός—a girdle.)

The Cestoda are all endoparasitic worms, and infect the intestinal canal of vertebrata.

General Characters.—The Cestodes differ from the Trematodes in being multiple in character. The tapeworm is not a single individual, but a multitude of organisms arranged in a chain, thus forming a compound jointed colony.

1. *Shape.*—The Cestoda are compound, flat, parasitic worms.

2. *Size.*—Varies much: some forms measure $\frac{1}{4}$ of an inch, others 24 feet in length.

3. *Structure.*—The adult worm or Strobila consists of a number of complete sexual individuals arranged in a chain. We have—

(1) The Head or Nurse, which is usually small in size, pyriform in shape, and has one or two suckers surrounded by a ring of chitinous hooklets, to enable the worm to cling to the intestines of its host. It has neither an alimentary system nor sexual organs.

(2) The Proglottides.—These are a series of segments produced one behind the other by a process of budding from the head or nurse. Each segment or proglottis resembles its neighbours except in size and degree of maturity. The segments farthest from the head are the oldest, the largest, and most mature; the segments next the head being immature and having no sexual organs. In an ordinary tapeworm there may be as many as 1200 of these segments.

Each proglottis has a complete water-vascular system, composed of parallel canals running on each side of the body, and united at the hinder end of each segment by cross branches. The proglottides have no digestive organs of any kind, being nourished by imbibition. They are hermaphrodite, and ova are formed by sexual union within the proglottides. A single proglottis may contain as many as 35,000 eggs.

Life History.—As above stated, the ova of the cestoda are produced in the proglottides, which when ripe break off from the rest of the chain and are cast out by the body of their host. Within these ripe proglottides the ova are already partially developed, and when ejected are full of active embryos. These embryos are enclosed in a membrane to protect them

from injury, and consist of a head furnished with three pairs of silicious spines or hooklets. By the decomposition of the proglottides the embryo-bearing ova are set free, reach water, and thence find their way into the stomach and intestines of their host.

The membrane enclosing the embryo is now ruptured mechanically, or digested by the gastric juice, and the embryos are liberated. They are called PROSCOLICES (*scolex*, a worm), and consist of a small vesicle with three pairs of silicious spines. By means of these hooklets the proscolex fastens itself to the intestinal wall, bores through it, and makes its way to the liver or other organ of its host. Here it becomes encysted, loses its hooks, and from its hinder end develops a small vesicle full of fluid.

It is now called a SCOLEX, which in some tæniada are known as *hydatids*, in others as *cysticercus*. When thus encysted, the scolex is composed of a vesicle united by a narrow neck to a head similar to that of the adult tapeworm, being armed with a circle of hooklets, and having four oscula or suckers. It has no reproductive system, nor, in fact, organs of any kind, and can undergo no further development unless it gains entrance into the intestinal canal of man or other host. This is effected by an animal eating flesh, etc., containing the scolices, when the cysts are digested and the scolices set free. They at once lose their caudal vesicle, attach themselves to the intestinal wall of their host but do not perforate it, and in this situation soon become the head of the future tapeworm and begin to produce proglottides which again pass through the cycle of development above described. Thus we have—

1. The *Ova* discharged from the ripe proglottis.
2. The *Proscolex*.—The minute embryo liberated from the ova when taken up from water, etc., by some animal.
3. The *Scolex*.—The more advanced, but still sexually immature, embryo into which the proscolex develops when it has become encysted in the tissues.

4. The *Strobila* or adult tapeworm, infesting the alimentary canal of its host, and composed of a head, neck, and proglottides.

Division.—The Cestodes which infest man are—

1. *Tæniada*—

- (1) *Tænia solium*.
- (2) *Tænia mediocanellata*.
- (3) *Tænia echinococcus*.

2. *Bothriocephalida*—*Bothriocephalus latus*.

I. *TÆNIA SOLIUM*.

Synonyms—*Tænia cucurbitina*, *Tænia humana armata*,
Tænia vulgaris.

Larva—Simple scolex, Measle, *Cysticercus cellulosæ*.

General Characters.—1. **SIZE.**—The adult worm or strobila measures about 2 to 10 feet long.

2. **SHAPE.**—It has a small head, long narrow neck, and transversely segmented body.

3. **STRUCTURE.**—

The *Head* is small, rounded, about the size of a pin-head, and consists of rostellum or beak with twenty-six hooklets, and of a wider part on which are four suckers.

Body.—Next the head comes a long, narrow, thread-like neck, followed by a series of larger segments—the proglottides. At first the segments are broader than they are long, and are immature—the remaining segments are the reverse, longer than broad, and are sexually mature proglottides. These proglottides are hermaphrodite, the genital orifices being placed alternately on each side of the body, and the male and female organs open by this common genital pore. They have a complete water-vascular system.

The *Ovary* consists of a central stem, with a number of lateral branches, each of which again branches. The testes appear as clear, white, convoluted tubes, with vesicles.

Ova are nearly spherical in shape, about $\frac{1}{50}$ inch in diameter, and are surrounded by a dense capsule which encloses the partly-developed, six-hooked embryos. These embryos give rise to the scolices in the flesh of the pig, the scolex in this case being called a "measle," or *cysticercus cellulosæ*, or bladder-worm.

Life History.—As above described.

Intermediate Host.—The pig, which gets the embryos from water—the scolices forming the "measles" of pork. Within these measles or cysts, the hooklets, which do not decompose, are often found after the scolex has perished. They are short, broad, hook-shaped bodies, with a small knob at their base.

Host.—Man; owing to eating imperfectly cooked, measly pork. It infects man, not only as the mature worm, but as *cysticerci*. The adult worm is found in man only.

Habitat.—The *immature* worm is found in subcutaneous tissues, muscle, brain, eye, liver of the pig; the *mature* form, in the small intestine of man.

II. TÆNIA MEDIOCANELLATA.

Synonyms—*Tænia saginata*, *Tænia dentata*, *Tænia inermis*,
Beef tapeworm.

Larva—*Cysticercus bovis*.

General Characters.—

1. **SIZE.**—Larger than *tænia solium*, both in length and breadth, often measuring from 14 to 24 feet. It is commoner in Great Britain than *tænia solium*.

2. STRUCTURE.—

The *Head* has four suckers, but no rostellum nor hooklets. Following the head is a narrow neck, and then the several segments or proglottides.

The *Ovaries* consist of many lateral processes, but these do not, as in the case of *tænia solium*, again branch, a character by which the proglottis of the one can be distinguished from that of the other.

Ova.—Similar to those of *tænia solium*.

Life History.—Similar to that of *tænia solium*.

Intermediate Host.—Cattle.

Host.—Man.

Habitat.—Immature form, in the muscles of cattle; as many as 300 having been found in a pound of flesh taken from psoas muscles.

The *mature* form occurs in the intestine of man.

III. BOTHRIOCEPHALUS LATUS.

Synonyms.—Broad tapeworm, *Tænia lata*, *Tænia grisea*,
Dibothrium latum.

General Characters.—

1. **SIZE**.—The largest known human tapeworm. It measures from 16 to 30 feet long, and about 1 inch broad, and consists of three or four thousand segments. It is very rare in Great Britain, but not uncommon in Central Europe and in Russia.

2. **STRUCTURE**.—The *Head* is small, oval, or club-shaped, with a longitudinal groove or slit on each side. It has no proboscis, nor suckers, nor hooklets.

The *Proglottides* are about 4000 in number, the largest being in the middle of the chain. They are each bi-sexual. The uterus consists of a simple, coiled-up tube, and the genital orifices are placed along the middle line of the ventral aspect—not on the sides, as in the last group.

The *Ova* are oval in shape, and about $\frac{1}{350}$ of an inch long. They have an operculum and a brown-coloured shell.

Life History. The ova are set free in the body of the host, and on reaching water are there hatched. The proglottides themselves are not discharged from the intestine as is the case with *tænia solium*. The embryo, which has a ciliated envelope, swims about in the water till the envelope bursts and liberates six-hooked embryos. These make their way into the muscles of some fresh-water fish, and there develop into the asexual larval worm.

If the fish be eaten by man, the larva develops into the sexual form above described.

Intermediate Host.—Certain fish, as pike, turbot, etc.

Host.—Man, dog.

Habitat.—Intestinal canal.

Effects.—Intestinal catarrh in children. In the adult the effects are very variable.

Distribution.—Germany, Russia, Poland, Sweden, Holland, Belgium, Ireland, England, France.

Symptoms due to any of these three worms :—

Frequently they give rise to no symptoms : there are in fact no pathognomonic symptoms ; but certain reflex disturbances are common, such as itching of the nose or anus, colicky pains, constipation alternating with diarrhœa, mental trouble, such as melancholia, epilepsy, etc., voracious appetite, and painless vomiting which may simulate brain disease. Anæmia, often of a severe type, has been frequently noted in connection with bothriocephalus. I have known all the symptoms of stone in the bladder to arise in a case dependent on reflex irritation from tapeworm. *If the above symptoms are present without any obvious cause or reason, always examine the fæces for segments of these worms.* A discharge of the segments is of course conclusive.

Treatment.—Having diagnosed the presence of the worm, then administer something which will paralyse the worm and afterwards expel it. The following treatment may be tried.—Give at night-time for six nights running, four grains of ipecac: with one grain of pil. hydrarg.; the seventh morning on an empty stomach give ʒj of the liquid extract of male fern suspended in mucilage, and four hours afterwards give half-an-ounce of Epsom salts and a drachm of ammon. chloride in a tumbler of effervescing water. I tried this course in eighteen consecutive cases of tapeworm, and in only two cases was the treatment unsuccessful in expelling the whole worm. Or, after keeping the patient on a fluid diet for twenty-four hours, give a

purgative at night, and next morning, after it has acted, give the male fern on an empty stomach ; 5ss may be safely given to a child, 5j-5ij to an adult. It is most easily taken in capsules, each containing 15 minims, one being given every ten minutes till the required dose is attained. The patient should lie quiet for some three hours to avoid nausea. A brisk purge is then given, and the worm is expelled. Turpentine, pumpkin seeds, etc., have their advocates.

Caution.—Be sure the head is expelled.

IV. TÆNIA ECHINOCOCCUS.

General Characters.—

1. SIZE.—*Tænia echinococcus* is a small worm, about $\frac{1}{8}$ to $\frac{1}{4}$ inch long.

2. STRUCTURE.—This worm consists of only four segments, including the head. The *Head* is pointed, has four suckers and a double circlet of hooks. These hooks are about 30 to 40 in number, and are shaped like those of *tænia solium*, but are much smaller.

The last proglottis, when mature, is equal in size to the rest of the body, and contains the reproductive organs. The genital pores are placed on the lateral aspect of the body. The ovaries are complicated, and the ova are small but not exceedingly numerous, and in them are developed the six-hooked embryos.

Life History.—When ripe the proglottides drop off and pass out of the body of the host—the dog or wolf. The embryos are now liberated on the ground, on plants, or in water, and thus gain access to the stomach of man. They then perforate the intestinal walls, and getting into the circulation, are by this or other means carried to the liver or other organ, where they become encysted and develop a spherical vesicle which may reach a great size. They are now called HYDATIDS.

Hydatid Cysts.—

1. STRUCTURE.—These cysts when fully formed are composed of three parts—

- (1) The false cyst formed by the tissues of the part.
- (2) The ectocyst—an opaque, chitinous membrane of great thickness, white in colour, smooth, glistening, and laminated.
- (3) The endocyst—a more opaque, granular layer, composed of nucleated cells, and covered by small white spots—brood capsules.

(4) Inside the cyst there is a colourless watery fluid which contains salts, but no albumin, a point of value in diagnosis.

Hydatid cysts are, moreover, much larger than those of *cysticercus cellulosæ*.

2. DEVELOPMENT.—Within these cysts the scolices—echinococcus heads—are developed in the following manner:—

On the inner wall of the cyst are formed small vesicles—*brood capsules*—which project into the cavity of the cyst. From the walls of these brood capsules small cup-like buds or hollows are formed, each of which gradually elongates and becomes a cæcum with its cavity opening outwards—*i.e.*, it communicates with the cavity of the brood capsule. Within these depressions or hollow buds the echinococcus head is developed, and, when mature, turns itself inside out—*i.e.*, everts itself, so that the head now projects into the brood capsule. These heads are similar to those of the adult worm, having a double circle of hooklets and four suckers.

Development cannot proceed further than this in the human body, but if the cysts gain access to the dog, etc., then the adult tapeworm is formed in the intestine.

SECONDARY CYSTS are often found in connection with the primary cyst. This may occur in one of three modes—

1. By a process of budding out of the wall of the ectocyst, thus giving rise to a number of daughter-cysts, side by side—*exogenous cysts*.

2. Again, the daughter-cysts may be formed inside the primary cyst—these are called *endogenous cysts*.

3. Or, the cysts may be *multilocular*—*i.e.*, composed of many separate alveoli divided from each other by dense fibrous tissue. They occur as hard, firm tumours in the liver.

Intermediate Host.—The cystic form is alone found in man.

Host.—The adult worm in the dog and wolf.

Habitat.—The *cystic* form is found in the liver, lungs, brain, heart, muscle; the *mature* form, in the intestine.

Diagnosis and Treatment.—See Hydatids of the Liver.

NEMATODA.

(Νῆμα—a thread).

Synonym.—Thread-worms or round worms.

General Characters.—The thread-worms are a very large and well-known group of helminths. They are simple, not compound, and do not form colonies. They closely resemble the common earth-worm, being round and thread-like without segmentation or appendages. They undergo no metamorphosis, the sexes are distinct, and there is a marked difference between the male and female—the male being smaller than the female.

STRUCTURE.—The Nematoda have a distinct alimentary canal with a mouth furnished with soft horny lips, an œsophagus, stomach, intestine, and anus. There is a thick elastic ectoderm or cuticle and a well-developed muscular system. The genital pore placed on the ventral aspect is, in the female, situated about the middle of its length; in the male, near the anus, where there is a chitinous prehensile investment.

The most common Nematoda are—

1. *Trichina spiralis*.
2. *Filaria sanguinis hominis*.
3. *Filaria medinensis*.
4. *Dochmius duodenalis* or *ankylostomum duodenale*.
5. *Ascaris lumbricoides*.
6. *Ascaris mystax*.
7. *Eustrongylus gigas*.
8. *Trichocephalus dispar*.
9. *Oxyuris vermicularis*.

I. TRICHINA SPIRALIS.

Synonyms.—Flesh worm, *Pseudalius trichina*.

Larva.—Muscle trichinæ, Encysted trichinæ, Flesh worms.

General Characters.—*Trichina spiralis* is a very minute worm, the male and female being distinct.

1. **SIZE.**—Male, $\frac{1}{18}$ inch ; female, $\frac{1}{8}$ inch long.

2. **STRUCTURE**—

The *Head* is narrow, pointed, unarmed, with a simple central oval aperture.

The *Body* is thread-like, bent upon itself, thicker behind than in front, and in both male and female the hinder part of the body is straight. In the male, however, it has a short, bilobed caudal appendage, between the lobes of which is the anus. The testes are convoluted tubes. The female is about $\frac{1}{8}$ of an inch long, rounder and shorter behind than the male. There is an ovary, vagina, uterus, and the genital orifice is near the head.

The *Ova* are $\frac{1}{170}$ inch long, and are hatched within the parent (ovoviviparous).

Larval Form.—the trichina of muscle—is a very small worm, about $\frac{1}{30}$ inch long, coiled up in a spiral manner within a fibrous capsule or cyst, the long axis of which lies in the long axis of the muscular bundles. A single capsule may contain two or more larvæ, and there may be as many as 325,000 of these capsules in an ounce of meat. They are especially common in the abdominal and thoracic muscles and appear as whitish spots from the cyst being often calcified towards the poles. This small worm has a digestive system and an imperfect sexual apparatus.

Life History.—When a piece of meat affected with trichinæ is eaten by an animal, the capsules are dissolved, and the embryo parasites which they contain are liberated. These mature in a day or two in the intestinal canal of the host. The sexes unite and give birth to ova and embryos ; a single ovum producing over 1000 embryos, and a single female discharging over 16,000 ova.

The embryos migrate from the intestine to the striped muscles, passing through the intestinal walls, but it is not clear how they reach the muscles—possibly through the peritoneal cavity or through the blood and lymph.

Once in the muscles the embryos penetrate the primitive bundles, reduce their contents to debris and soon become mature muscular trichinæ, forming cysts, part of which is made up of a chitinous secretion of the parasite, part of a wall of fibrous tissue formed of the perimysium of the muscle bundles. These cysts, as above stated, may become partly calcareous, giving rise to white shining spots in the muscle. They may remain quiescent for years.

Intermediate Host.—The trichinæ are found in pigs, rabbits, sheep, dogs, rats, mice. The pig gets them from the rat, which acquires them from human fæces.

Host.—Man, finding their way into his body through eating uncooked pork.

Habitat.—The adult worm only inhabits the intestine and only lives for a few weeks.

Symptoms of Trichiniasis.—Unless there be a large number of embryos eaten definite symptoms may not arise ; but in well-marked cases of trichiniasis the symptoms are very characteristic. A few hours or days after eating the infected flesh symptoms of gastro-intestinal irritation appear ; there may be vomiting, diarrhœa, and abdominal pain.

Towards the second week great soreness and stiffness of muscles develop, the temperature runs up, and may be remarkably remittent in character, a characteristic œdema sets in at first in the face, but becomes specially marked in the affected muscles. In protracted cases the patient becomes emaciated, exhausted, and a typhoid condition may supervene and carry off the sufferer.

There is marked leucocytosis, the eosinophil cells being specially preponderant.

It will be easily understood that the more important

muscles such as the diaphragm may be early implicated, and a fatal issue is then speedily brought about.

The disease occurs in epidemic form in some countries.

Diagnosis.—Examine stools, or pieces of muscle that have been harpooned. The more important diagnostic features are—

1. Severe pains in the joints without marked swelling.
2. Edema.
3. Dyspnoea.
4. Marked muscular pain.

Prophylaxis.—All meat to be thoroughly well done. Avoidance of pork in an infected district.

Treatment.—Purgatives at first, combined with twenty grains of salol every night. Later, give five grains of quinine suspended in two drachms of glycerine thrice daily. Treat symptoms as they arise. No specific remedy. Morphia may be required in large doses to relieve the intolerable pain.

II. FILARIA SANGUINIS HOMINIS.

Synonyms—Filaria Bancrofti, Filaria cystica, Trichina cystica, Filaria nocturna.

General Characters.—The mature forms are rarely seen. The sexes are distinct. The male is smaller than the female, and lives in the same vessels.

“The female is described as a small slender hair-like worm with a club-shaped head, a narrow alimentary canal, a two-horned uterus usually full of embryos. These are discharged through the vagina, which opens near the mouth.”

The embryos measure about $\frac{1}{10}$ inch long, have a rounded head, a tapering tail, and are enclosed in a fine membrane which does not burst, but which elongates as the embryo uncoils itself, thus forming a delicate sheath to the embryo.

Life History.—The adult worms inhabit the lymphatics. The embryos may sometimes gain access to the stomach of their host through drinking-water; they thence make their way to the vessels. Far more often the embryos are sucked

up by the female mosquito from the infected blood. In the stomach of the mosquito they cast their sheath, become actively mobile, and pass into the thoracic muscles. There they grow for about a week, again become mobile, and pass to the base of the proboscis, from which they enter the blood of the next person bitten, and complete their development in man. Here the sexes unite, and the embryos are discharged into the lymphatics. In the daytime the embryos are found in the lymphatics, but at night they crowd the blood stream. They cannot, however, undergo further development in man, and hence are taken up by a species of mosquito, in the body of which the embryos are matured. When the insect dies they find their way to water, thence to the stomach of their host, and, on reaching the blood and lymphatic system, form the mature filaria.

Intermediate Host.—Mosquito.

Host.—Man.

Habitat.—The embryo, called *filaria sanguinis hominis* or *filaria nocturna*—in the blood and urine; the mature form, called *filaria Bancrofti*—in the lymphatics.

Symptoms.—The passage of an opaque milky urine tinged with blood is the most prominent symptom in the bulk of cases; *but the enormous lymph scrotum and certain forms of elephantiasis are also due to the presence of this parasite.* Sometimes a blood-clot may form either in the bladder or pelvis of the kidney giving rise to troublesome symptoms.

Treatment.—At present very unsatisfactory. Iodide of potassium, turpentine, carbolic acid, etc., have their advocates.

III. ANKYLOSTOMUM DUODENALE.

A short, white, cylindrical worm, found in the upper part of the small intestine. Female $\frac{1}{2}$ inch, male $\frac{1}{3}$ inch long. The mouth has four strong hooks and two conical teeth. Ova are very numerous, and may be found in the fæces. The larval stage is passed in water or damp earth. The parasite is

common in the tropics, especially Egypt, but is found also in Europe, and sometimes in this country, principally among miners.

Symptoms.—The worm sucks blood from the intestinal mucosa. If there are many of them this may lead to profound anæmia, epigastric pain, diarrhœa, dyspnœa and dropsy, and even to death. Many of the workers in the St. Gotthard tunnel died of ankylostomiasis.

Treatment.—All drinking-water should be boiled in districts where the parasite is found. Keep the patient on fluid food for a few days, then give 20 to 30 grains of thymol in cachet, and repeat two hours afterwards. Give a purge three hours later. After it has acted the patient may return to more solid food. Avoid alcohol, as it dissolves thymol, and may cause poisoning. Repeat the treatment once a week, so long as ova are found in the fæces.

IV. ASCARIS LUMBRICOIDES.

Synonyms.—Round-worm, *Lumbricus teres hominis*.

General Characters.—These parasites closely resemble the common earthworm.

SIZE.—The male measures about 4 to 6 inches; the female, 10 to 16 inches long.

STRUCTURE.—This parasite is a broad, smooth, fusiform, translucent, brown or red coloured worm, with fine circular striæ. Its anterior extremity has a three-lobed mouth. The tail is bluntly curved in the male, and has a double spicule near its end.

Ova, oval-shaped, $\frac{1}{300}$ to $\frac{1}{150}$ inch in diameter, and have a hard shell and an albuminous envelope.

Life History.—Not fully known.

Host.—Man, pig.

Intermediate Host.—Not required.

Habitat.—The ileum, colon, also mouth and nose. They are passed by the fæces, or are vomited.

Symptoms.—Very indefinite, in fact there are no characteristic symptoms. Picking of the nose, grinding the teeth, foul breath, etc., are the popular symptoms of the presence of this worm, but obviously any irritation of the intestinal canal may cause such symptoms. Perhaps we regard the presence of worms as a source of peripheral and intestinal irritation too lightly at present. Our forefathers distinguished a “worm fever” and prescribed successfully for that ailment.

Treatment.—Santonin four grains followed by three grains of calomel.

OXYURIS VERMICULARIS.

Synonym—Thread-worm.

Characters.—Female thread-like, with tapering tail, $\frac{3}{8}$ inch long. Male $\frac{1}{8}$ inch, tail curled. Ova are numerous, each containing a partly formed embryo. They are expelled with the fæces. The embryos develop fully when the ova are taken into the stomach, becoming adult in the small intestine. After conjunction the male dies, and the impregnated female passes into the colon and rectum. The worms often wriggle out at the anus, causing irritation; the patient scratches, and may thus transfer ova on his fingers to his mouth, and so reinfect himself.

Symptoms.—Presence of the parasite in the stools, irritation about anus and genitals.

Treatment.—Clear out lower bowel by enema, then inject by the long rectal tube Inf. Quassia, salt solution ʒij to Oj, or Tr. Fer. Perchlor. ʒij to Oj. From 10 to 20 ounces may be introduced, and retained as long as possible. Hands, nails, and perineum must be kept very clean, and carbolic lotion applied daily.

TREMATODA.

(Τρῆμα—a hole).

Synonyms—Flukes, Flat-worms, Suctorial-worms.

The only parasite under this class that we shall consider is

DISTOMUM HÆMATOBIUM.

Synonyms—Bilharzia Hæmatobia, Thecosoma, Schistosoma, Gynæcophorus.

General Characters.—They are dioecious, the sexes being distinct.

1. MALE—

(1) **SIZE.**—About $\frac{1}{2}$ inch long.

(2) **SHAPE.**—Cylindrical, with a canal or groove—gynophoric canal—at the posterior end of the body, in which the female is lodged.

2. FEMALE—

(1) **SIZE.**— $\frac{4}{5}$ inch long.

(2) **SHAPE.**—Thread-like.

Both male and female have two ventral suckers, and the reproductive orifice is below the ventral sucker.

Ova.—Oval in form, $\frac{1}{180}$ to $\frac{1}{160}$ inch in diameter, with a spine at the ends or at the sides of each egg.

Life History.—Not known. The larval forms are common in rivers and canals.

Intermediate Host.—Not known.

Host.—Man and monkey; got by drinking-water.

Habitat.—The blood. Is especially found in the inferior vena cava and portal veins, and in the vesical and hæmorrhoidal veins.

Effects.—The sexes unite in the blood—the ova are discharged, and pass through the walls of the bladder and ureter by means of the ulcerated surfaces caused by the parents. If

in large numbers they give rise to inflammation and hæmorrhage from the affected mucous membrane, causing endemic hæmaturia—or if the large intestine be affected, a special form of diarrhœa is the result. The ova pass out of the body by these channels, and can be found in the urine of the patient. If placed in warm water, the ova give rise to the free ciliated embryos.

Distribution.—This parasite is rarely met with in England, but is common in Egypt, Cape, Natal, also in Brazil.

DISEASES OF THE LIVER.

BEFORE attempting the study of diseases of the liver and kidney, the reader is earnestly advised to recall to mind the chief physiological facts regarding their functions and *relations to each other*. It will be remembered that the kidney is to a large extent the hand-servant of the liver; it excretes the waste material formed by the former, and thus enables the liver to perform its duties efficiently, and, at the same time, keep the blood free from morbid products. But it must also be understood that an alteration in their perfect physiological relation may be disturbed by—

1. The liver putting excessive strain on the kidney ; or,
2. The kidney, through disease, being unable to perform its normal functions, and thus clogging the liver.

FUNCTIONS OF THE LIVER

Will be considered briefly under three heads—

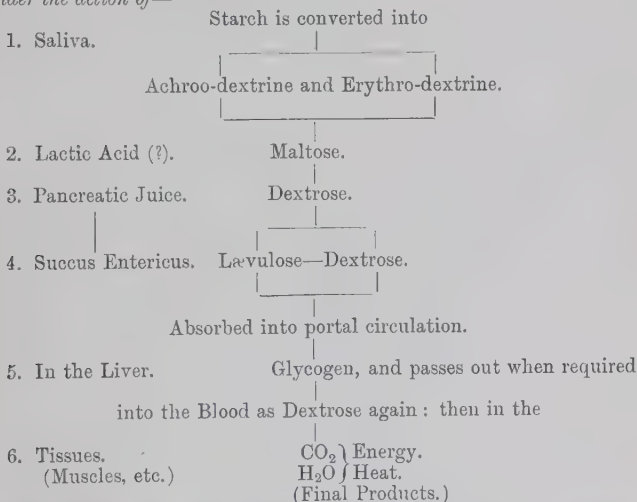
1. The metabolism of carbohydrates.
2. The metabolism of proteid material.
3. Bile formation.

If we study these carefully, though briefly, it will be easy for even a beginner to understand those facts which are necessary, in order to get a grasp of “*how*” such conditions as gout, diabetes, Bright’s disease, jaundice, and so on, are brought about. I must, however, warn the student that though the following statements represent our present knowledge of such metabolic

changes, they must be regarded as partly hypothetical, though sanctioned by the latest research.

I. METABOLISM OF CARBOHYDRATES.

Under the action of—



We see by the above table that carbohydrates, after passing through various changes, get to the liver as grape sugar. In the liver a portion is, however, stored up for a time, *not as sugar*, but as a *peculiar starch, termed glycogen*. The amount of glycogen which can be obtained by extraction from the liver varies with the nature of the diet. Working with dried dog's livers Pavy found the following results,—after animal food, 7·19 per cent.; after animal food with sugar, 14·15 per cent.; after a vegetable diet, 17·23 per cent. Thus the formation of glycogen is favoured by a free supply of carbohydrates, dextrose in particular, though lævulose is also used. But the liver also forms glycogen from proteid, though not to the same extent. It forms no glycogen from fat.

What becomes of the glycogen? Bernard considered that the liver simply formed it and kept it stored up, to prevent

flooding of the organism with dextrose, and gave it out again to the tissues, retransformed into dextrose, when the amount of dextrose in the blood had fallen below a definite minimum. Pavy's view (the more modern) is that dextrose is not used in the body as such, but combined with proteids, and that the liver simply stores the sugar as glycogen until it has time to synthesise it into a form suitable for the tissues. Extensive formation of dextrose from glycogen is thus pathological, and only occurs in diseases such as diabetes. The blood then is surcharged with sugar; the muscles say, "We do not want so much": there is an increase of the supply over the demand—"a glut in the market," as it were, and the sugar is excreted by the kidneys, and temporary *glycosuria* results. But supposing the excessive formation of dextrose from the glycogen of the liver is due to some permanent neurosis, organic nervous disease, or other general condition, then we should get *glycosuria plus wasting of muscles*, or in other words, *diabetes*. We thus see that *glycosuria* is a temporary excessive supply; *diabetes*, on the other hand, is a serious organic disease. But we must remember that a mere excess of sugar persistently floating in the blood must act as foreign matter and induce changes in the tissues by its irritating action; so a continued *glycosuria* may induce real *diabetes*. Other theories of *diabetes* have been discussed under that disease.

II. PROTEID METABOLISM.

Proteids, after being subjected to the various digestive agents, saliva (mastication), gastric juice, pancreatic juice, and succus entericus, are finally absorbed into the portal circulation; carried to the liver, and there broken up or divided into (1) That which is to be used for building up the tissues (see above); (2) That which is to be excreted. That which is excreted goes through many changes before it can be eliminated *via* the bile and urine. Graphically we might represent such changes as follows.

Proteids = substances of an albuminous nature, rich in nitrogen. *Under the action of—*

1. Saliva,—they are finely divided or masticated ;
2. Under the action of gastric juice they are converted into—

(1) Albuminates (acid).	} = Acid chyme.
(2) Albumoses (many kinds).	
(3) Peptones.	

3. Under the action of pancreatic juice and succus entericus they are converted into—

- (1) Alkali albuminates.
- (2) *Albumoses*.
- (3) Peptones.
- (4) Leucin, tyrosin—to a slight extent.

Skatol, indol, and phenol—*bodies which result from putrefaction* (of course under bacterial influence) are also formed, especially if absorption be delayed.

Proteids are thus (if fully digested) converted from non-diffusible into diffusible bodies termed “peptones,” with the addition of some *putrefactive bodies* (which ordinarily should be excreted *via* the fæces). We thus see even at this stage how powerfully constipation can affect the system, by *increasing the formation* of these poisonous products, and by the same delay increasing the chance of their absorption.

The ultimate result of the action of the liver in dealing with nitrogenous waste products is the formation of urea. Possibly some small proportion of urea is derived from the nitrogenous waste of every body tissue, but the main source of its formation is undoubtedly the liver. If the liver of a frog be removed, the formation of urea almost ceases. In mammals, if the liver be thrown out of action by joining the portal vein to the inferior cava, urea is much diminished. In both cases its place is taken by ammonia: in both, if a proteid meal be given, ammonia poisoning appears, and excess of ammonia is found in the blood. In birds uric acid is the chief nitrogenous excretion in the urine. In them extirpation of the liver means disappearance of uric acid and appearance of ammonia. The liver therefore forms urea from the ammonium compounds circulating in the

blood. It has also been demonstrated that it can form it from the putrefactive bodies above referred to—glycine, leucine, etc.

Compare these experimental results with the results of clinical examination of the urine in diseases of the liver. In cirrhosis there is marked diminution in the amount of urea, and increase of ammonia. In acute yellow atrophy, and also in phosphorus poisoning, the urea is enormously diminished, and leucine, tyrosine, and ammonia take its place. *Failure on the part of the liver to do its work results in imperfect oxidation of waste products*, the nature of the products so formed depending on the degree of oxidation. Comparatively slight defect may be attended by excess of uric acid (gout). If the defect be greater there is excess of ammonia and its compounds in the blood and urine, and where the hepatic function is almost abolished, leucine, tyrosine, etc., appear unchanged.

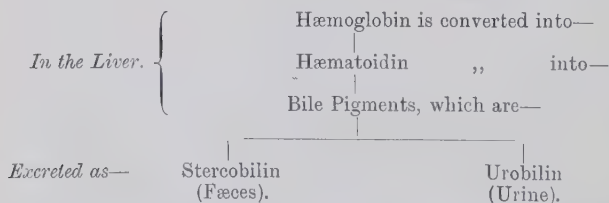
III. BILE FORMATION.

Bile consists principally of water, cholesterin, bile salts, bile pigments, and mucin. It is to be regarded as—

- (1) A secretion—concerned in the proper assimilation of fats, etc.
- (2) An *excretion*—a means by which broken-down blood pigments are excreted, with also a certain amount of proteid waste.

Bile pigments and bile salts are formed only in the liver; cholesterin, lecithin, etc., exist as such in the blood.

FATE OF BLOOD PIGMENTS.



The pigment of bile being derived from hæmoglobin, *per se*, we must have a larger amount of bile pigment in those

diseases attended with the *destruction of red corpuscles*. Now bile pigment increases the thickness of bile, and Quincke has shown, "When there is great destruction of red corpuscles in the liver, mucin is also increased, which in turn materially increases the viscosity of bile." Thus in pernicious anæmia the bile is rendered thicker by—

1. Increased pigment.
2. Increased mucin.

If we increase the thickness of a fluid running through a tube, we do two things—

1. Retard the flow.
2. Cause a greater tension of the tubal walls.

Now, remembering all this takes place in the liver in all exhausting diseases, it is no wonder we get a sallow complexion; for the bile tension being higher than normal, there is every facility for transudation of bile into the blood. This is well seen in pernicious anæmia. *Probably all cases of jaundice are, in a sense, obstructive*, not indeed through *actual stoppage* or suppression of bile, but through the increased tension within the bile ducts. Brunton has done much to throw light on this subject. Lastly, in summing up the functions of the liver, we must remember exercise and moderate eating and drinking must increase its functional activity; then the bile moves on more quickly, glycogen is properly stored up, and though more is formed than usual, it does not accumulate, as the muscles use up its synthetic products more rapidly to furnish fuel for their increased work. *Urates decrease and urea increases*. The reverse must happen by sluggish habits and intemperance. *Local vascular dilatation* as a result of disease may also *increase for a time the functional activity*, but soon venous congestion would occur under such circumstances, and torpidity succeed the activity.

JAUNDICE.

Jaundice is the name applied to a group of symptoms, arising from the circulation of bile in the blood. We have already pointed out that bile is to be looked upon as a

secretion and also as an excretion. Its retention within the liver and its absorption into the blood must therefore give rise to symptoms due to—

1. *Its absence from the intestines.*—This interferes largely with the perfect assimilation of fat, and the imperfect absorption causes serious decomposition or putrefactive changes to take place in the intestines, skatol and indol are formed in excessive quantities, *complete* proteid digestion cannot go on, and thus we get absorption of albumoses and other poisonous compounds, which contribute largely to the serious blood changes to be next described.

2. *The circulation of bile within the blood* causes destruction of red corpuscles and, therefore, anæmia; bile is also a neuromuscular poison, paralysing both nerve centres and muscular fibres; consequently we get imperfect action of the heart, slow pulse, depression of the spirits, mental torpidity, and possibly profound coma. Two types of jaundice are described—viz., obstructive and non-obstructive.

In the former case no bile enters the intestine, and the seats of obstruction are probably in the large ducts. By non-obstructive jaundice is meant a condition of icterus in which a certain amount of bile escapes into the intestine. We have already shown how the increased viscosity of bile, resulting from excessive breaking down of red corpuscles, might raise the bile pressure sufficiently to cause absorption of bile into the blood, and it is highly probable that all the so-called cases of non-obstructive jaundice are due to (a) increased destruction of blood with increased supply of hæmoglobin to the liver, and (b) the action of poisons, bacterial or other. This class of cases is therefore spoken of as Toxæmic Jaundice. It must be remembered, however, that after *jaundice due to obstruction* has existed for a time, the distended bile ducts may obliterate or obstruct the *vascular* supply to the hepatic lobules, and thus cause true suppression of bile formation. Obviously the symptoms of jaundice are not due to actual suppression, but to the causes which lead to such abolition of the biliary function. I append Dr. Murchison's tabulated causes of jaundice.

A.—*Jaundice from Mechanical Obstruction of the Bile Duct.*

- I. OBSTRUCTION BY FOREIGN BODIES WITHIN THE DUCT.
 1. Gall-stones and inspissated bile.
 2. Hydatids and distomata.
 3. Foreign bodies from the intestines.
- II. OBSTRUCTION BY INFLAMMATORY TUMEFACATION OF THE DUODENUM OR OF THE LINING MEMBRANE OF THE DUCT, WITH EXUDATION INTO ITS INTERIOR ("Catarrhal Jaundice").
- III. OBSTRUCTION BY STRICTURE OR OBLITERATION OF THE DUCT.
 1. Congenital deficiency of the duct.
 2. Stricture from perihepatitis.
 3. Closure of orifice of duct in consequence of an ulcer in the duodenum.
 4. Stricture from cicatrisation of ulcers in the bile ducts.
 5. Spasmodic stricture.
- IV. OBSTRUCTION BY TUMOURS CLOSING THE ORIFICE OF THE DUCT, OR GROWING IN ITS INTERIOR.
- V. OBSTRUCTION BY PRESSURE ON THE DUCT FROM WITHOUT, BY—
 1. Tumours projecting from the liver itself.
 2. Enlarged glands in the fissure of the liver.
 3. Tumour of the stomach.
 4. Tumour of the pancreas.
 5. Tumour of the kidney.
 6. Postperitoneal or omental tumour.
 7. An abdominal aneurism.
 8. Accumulation of fæces in the bowels.
 9. A pregnant uterus.
 10. Ovarian and uterine tumours.

B.—*Jaundice independent of Mechanical Obstruction of the Bile Duct.*

- I. POISONS IN THE BLOOD INTERFERING WITH THE NORMAL METAMORPHOSIS OF BILE (TOXÆMIC JAUNDICE).
 1. The poisons of the various specific fevers.
 - (a) Yellow fever. (b) Remittent and intermittent fevers.
 - (c) Relapsing fever. (d) Typhus. (e) Enteric fever.
 - (f) Scarlatina. (g) "Epidemic jaundice."
 2. Animal poisons.
 - (a) Pyæmia. (b) Snake-poison.
 3. Mineral Poisons.
 - (a) Phosphorus. (b) Mercury. (c) Copper.
 - (d) Antimony, etc.
 4. Chloroform or ether.
 5. Acute atrophy of the liver.

II. IMPAIRED OR DERANGED INNERVATION INTERFERING WITH THE NORMAL METAMORPHOSIS OF BILE.

1. Severe mental emotions, fright, anxiety, etc.
2. Concussion of the brain.

III. DEFICIENT OXYGENATION OF THE BLOOD INTERFERING WITH THE NORMAL METAMORPHOSIS OF BILE.

IV. EXCESSIVE SECRETION OF BILE, MORE OF WHICH IS ABSORBED THAN CAN UNDERGO THE NORMAL METAMORPHOSIS.

1. Congestion of the liver.
- (a) Mechanical. (b) Active. (c) Passive.

V. UNDUE ABSORPTION OF BILE INTO THE BLOOD FROM HABITUAL OR PROTRACTED CONSTIPATION.

Symptoms of Jaundice—

1. Icterus or tinting of the skin, conjunctivæ, secretions, etc. The colour ranges considerably, from a lemon-yellow to a deep greenish black (black jaundice). Yellow vision or xanthopsia is sometimes present.

2. Gastric disturbances, flatulence, nausea, and often complete anorexia.

3. Constipation, often alternating with diarrhœa; the fæces are pale, intensely foetid and pasty in character.

4. Slow pulse (has been observed in some cases to number only 20 per minute).

5. Extravasation of blood.

6. Cerebral symptoms. Marked depression of spirits, "patient sees things with a jaundiced eye," melancholia, and, in the graver forms, an assumption of the typhoid state ending in death.

7. Itchiness of the skin.

The tests for bile acids and bile pigments will be found under Examination of Urine.

Treatment.—As jaundice is merely symptomatic, the cause must be treated. In the milder forms a catarrh of the alimentary canal, or the presence of small biliary calculi, will require attention. For detailed treatment see Hepatic Diseases and Gall-Stones. Remember, however, the indications always are to—

1. Remove obstruction *if possible*.
2. *Promote functional activity by hepatic stimulants, after clearing old bile away with saline purges, calomel, or aperient waters.*

ICTERUS NEONATORUM.

The form of jaundice which occurs amongst new-born infants may be of either a mild or a malignant type. The mild form appears on the second or the third day, and lasts from seven to fourteen days; beyond the pigmentation few symptoms are present. There may be bile in the urine, and the fæces are pale.

Causes.—

1. The jaundice is possibly due to the large destruction of red corpuscles, which takes place the first few days after birth.
2. Patency of the ductus venosus, allowing the portal blood (which contains at this time bile pigment) to mix with the systemic circulation.
3. Diminished pressure in the portal circulation after severance of the placenta; the bile-pressure may then exceed the blood-pressure.

The etiology is, however, very obscure.

Malignant Form.—Causes.—

1. Congenital absence of the hepatic duct.
2. Congenital syphilitic hepatitis.
3. Phlebitis of the umbilical vein.

This form is invariably fatal.

ACUTE YELLOW ATROPHY OF THE LIVER.

(FATAL JAUNDICE.)

A peculiar and rare disease due to some unknown poison, and characterised by a rapid and often complete destruction of the hepatic cells throughout the gland.

Ætiology.—Women are more often attacked, and pregnancy is sometimes associated with the condition. Other causes put down are abuse of mercury, fright, the poisons of malaria, and typhus. (Though there are many points of resemblance between acute atrophy, yellow fever, and phosphorus poisoning, the conditions of each are not identical.)

Pathology.—The liver is much diminished in size, soft and flaccid. The capsule is wrinkled. On section, destruction of hepatic cells, and empty bile ducts, are the marked features. The liver contains little fat. Masses of golden pigment, red patches, and coarse granules are often present, *leucin and tyrosin can always be detected, and may crystallise spontaneously after death.*

Symptoms.—Usually there are precursory symptoms such as mental and bodily depression, constipation, and tenderness in the hepatic region. Slight simple jaundice may precede the fully developed disease. Mild fever is usual; in some cases it may run up to 106°. In the confirmed stage, intense jaundice sets in, with vomiting, etc., severe hæmorrhages, evacuations of pale fæces (or the fæces may be black from mixture of blood); pregnant women abort, and the typical typhoid state ushers in the end. The liver dulness gradually diminishes, and may ultimately disappear. Death usually takes place within seven days of the confirmed stage.

Diagnosis.—*The severity of the symptoms, with the jaundiced aspect, diminished hepatic dulness, and the presence of leucin and tyrosin in the urine, make the diagnosis fairly easy.*

We have already seen that leucin and tyrosin are probably the earliest antecedents of urea. Their appearance in the urine, and disappearance of urea in a condition where the liver is so largely affected, forms a strong link in the chain of evidence in

favour of the liver being the chief seat of proteid metabolism, and urea the final product of such metabolism.

Treatment.—Probably useless. Large doses of quinine with the mineral acids, and diffusible stimulants may be tried.

ABSCESSSES IN THE LIVER.

Suppuration in the liver may be due to many causes, such as—

1. Suppurative pylephlebitis.
2. Tropical abscess, usually single.
3. Pyæmic abscesses, usually multiple.

I. SUPPURATIVE PYLEPHLEBITIS.

This disease follows upon suppuration in the portal area consecutive to diseases of the intestinal tract, and most often of the large intestine. It may follow suppuration of the mesenteric glands, or abscess of the gall bladder. The liver is enlarged and congested. There may be cloudy swelling of the liver-cells. On the surface, and extending through the substance of the organ, are branching yellow areas, due to suppuration in the walls of the veins.

II. TROPICAL ABSCESS

Is due to the amœba coli or to a bacterium, possibly the bacillus coli, flourishing in a suitable soil furnished by dysentery, excessive use of alcohol in hot climates, etc.

Pathology.—The abscess is usually single, but there may be two or more.

Situation.—Right lobe.

Walls.—Firm and thick, and have often three distinct layers—

1. *Inner.*—Grey in colour, and composed of necrosed tissue, amœbæ, and pus cells—no membrane internally.
2. *Middle.*—Brownish-red friable liver tissue.
3. *Outer.*—Hyperæmic liver tissue, surrounded by connective tissue.

Contents.—In amœbic abscess, amœbæ and detritus of necrosed liver tissue; in bacterial abscess, pus, often like anchovy sauce, and of a peculiar disgusting, sourish odour. The abscess if left alone most often bursts *into the right lung*.

Symptoms.—The *earlier* symptoms are—

1. Pain, sense of weight and fulness, and tenderness in the right hypochondrium.
2. More or less jaundice.

Later.—We get rigors, elevation of temperature, “septic” in character; symptoms dependent on more or less peritonitis; the tongue becomes furred and dry, a sickly odour emanates from the breath; jaundice may become marked, although this is uncommon, and ascites may develop. The physical signs are irregular enlargement of the liver, increase in the area of hepatic dulness both upward and downward, downward displacement of the hepatic margin, sometimes a rounded smooth prominence in the right hypochondrium, and local tenderness (not always present). It must be remembered that the symptoms may be latent until those of rupture appear. *When the abscess bursts into the lungs, the expectoration of anchovy paste-like sputum is characteristic.* If there be doubt about the diagnosis, puncture with a fine trocar. This should not be done unless, if pus be found, the major operation is intended to follow at once. The exploration may be repeated if pus is not found at the first attempt.

Treatment.—Avoidance of alcohol; removal from tropical climate; treat on general principles. Rest, ipecac., warmth, and tonics. When pus has formed and can be fairly localised, the abscess must be opened. For details see a work on surgery.

PYÆMIC ABSCESES.

Pyæmic abscesses of the liver are usually small and scattered throughout the liver substance.

Pathology.—The condition is essentially dependent on the

presence of micrococci, and is most frequently a part of a general pyæmia. The micrococci may gain an entrance into the liver substance either—

1. *Through the hepatic artery* in cases of general pyæmia, especially after head injuries (in which case the organisms must have first passed through the capillaries of the lung).

2. *Through the portal vein* in lesions of the portal area.

3. *Through the bile ducts* in ulceration from gall-stones, etc. In these cases the suppuration is in the course of the distribution of the portal vein. (Dr. F. TAYLOR.)

The formation of pyæmic abscesses has already been described under Pyæmia.

Symptoms.—Hectic fever, jaundice, with a tender, painful, regularly enlarged liver, are the main symptoms. Often the symptoms are very obscure, and pain may be conspicuous by its absence. “Febrile jaundice” is a valuable diagnostic indication when present.

Treatment.—Almost hopeless. Can only hope to support patient until the septic processes have ceased.

CIRRHOSIS OF THE LIVER.

This is a condition characterised by a great increase of connective tissue at the expense of the proper glandular tissue. It occurs in two forms (1) multilobular, atrophic, or alcoholic cirrhosis, and (2) unilobular, hypertrophic, or biliary cirrhosis. Intermediate stages exist, and the two forms are not very sharply separable.

Ætiology.—Hepatic cirrhosis results from poisons reaching the liver either by the alimentary canal, and hence through the portal vein, or else by the hepatic artery. Of those causes that may affect the liver by way of the portal vein, alcohol, especially in the form of spirits, is certainly the most important. The disease is therefore most common in the middle period of life, although cirrhosis may occur in childhood from early

intemperance. Other causes may be found in dietetic faults, as in the excessive use of hot condiments. It is possible that toxins or micro-organisms reaching the liver by the hepatic artery may cause cirrhosis, but this is not definitely proved. Association with congenital syphilis has been occasionally noted. Cirrhosis is sometimes found as a complication of tuberculosis and malaria.

Morbid Anatomy.—During the hypertrophic stage, the liver is often enormous in size, extending even *below* the umbilicus; towards the latter end of the atrophic stage the liver may only weigh two pounds, and be quite inaccessible to the touch. The more atrophied the liver, the more rough, nodular, or hobnailed is it likely to be. Conversely, the larger the liver the greater probability of its being smooth or but slightly roughened. Cirrhosis of the liver is essentially a chronic inflammation of the connective tissue in the portal areas, leading to secondary degeneration and atrophy of the hepatic cells. There are therefore—(1) a stage of hypertrophy, and (2) a stage of atrophy. The view that cirrhosis is due to a primary degeneration of the liver cells, and that the fibrosis is due to replacement, is not so widely held.

Two forms are described—(1) ordinary or multilobular cirrhosis, (2) “biliary” or “monolobular cirrhosis.”

Dr. Woodhead’s table of the “biliary” and ordinary cirrhosis is appended :—

COMMON OR MULTILOBULAR
CIRRHOSIS.

1. The bile ducts are not involved in the growth of connective tissue at first; consequently, jaundice is a late symptom.

2. The new growth involving the portal circulation is likely to cause ascites, hæmorrhoids, hæmatemeses, etc.

BILIARY OR MONOLOBULAR
CIRRHOSIS (HANOT’S DISEASE).

1. The bile ducts are involved early, and the jaundice is a severe symptom. Apparently there is a new formation of bile ducts. [More probably these are the remains of the displaced ducts of the lobules.]

2. The portal veins are not involved, and symptoms dependent on portal obstruction are rare.

COMMON OR MULTILOBULAR
CIRRHOSIS.

3. In the earlier stages the increased growth of young connective tissue in the portal spaces causes considerable increase in the size of the organ; but in the later stages this tissue contracts, and in contracting causes a considerable decrease in the size of the liver.

4. The liver rough and hobnailed on its surface; and the capsule much thickened.

5. The masses of liver-cells vary in size, some consisting of several lobules, whilst others are smaller than a lobule. Each of these masses forms a *distinct area* having a rounded outline surrounded by a fibrous zone, and from the fibrous mass the mass of liver-cells can be easily turned out.

6. On microscopic examination it is seen that the process is going on *chiefly* at the periphery of the lobules, but that groups of lobules are affected.

BILIARY OR MONOLOBULAR
CIRRHOSIS (HANOT'S DISEASE).

3. The large amount of new tissue diffused throughout the organ causes a great enlargement of the liver.

4. Surface is smooth (like morocco leather); the capsule is not thickened.

5. The masses of liver-cells consist of atrophied lobules, and the cut surface has a more or less uniform, and finely granulated appearance.

6. The single lobules above mentioned are surrounded by bands of fibrous tissue, which bands, however, are not confined to the periphery, but invade the substance of the lobules.

The main thing to remember is *not* the varieties, but to look upon cirrhosis pathologically as a condition characterised by—

1. Increased growth of fibrous tissue in Glisson's capsule, and in the prolongations of it running into the liver substance.
 2. Obstruction to the portal circulation.
 3. Increased blood-pressure in the hepatic arteries.
 4. Obliteration of hepatic cells
 5. Obstruction of biliary ducts
- } *later.*

Symptoms.—At first there may be little disturbance

beyond symptoms of chronic gastric catarrh—viz., morning vomiting, anorexia, and acid eructations, but soon a severe hæmatemesis or œsophageal hæmorrhage coupled with a distinct sense of weight in the hepatic region may reveal the true nature of the disease, and on examination the liver is found to be much enlarged. Hæmorrhoids are very commonly present. The general symptoms are emaciation, pallor, and marked abdominal enlargement. The characters of the enlarged liver are that its surface is firm and free from tenderness, and its edge hard and sharp. If there is much emaciation the granulations may be felt on the hepatic surface ("hob-nailed liver"). The gall-bladder is not enlarged, but the spleen is, owing to portal obstruction. As time wears on the more characteristic features are—

1. Ascites.
2. Emaciation.
3. Jaundice (often very late).

And now on examination the liver is found atrophied.

The ascites as a rule is particularly well marked. The surface of the abdomen is covered by large distended veins, showing an attempt at collateral circulation to carry on the now obstructed portal circulation. This condition is highly important from a prognostical point of view. The more common anastomoses are between—

1. The gastric and œsophageal veins.
2. Middle hæmorrhoidal of the inferior mesenteric and inferior hæmorrhoidal of the internal iliac vein.
3. Coronary veins of stomach and phrenic veins.
4. Accessory portal vein of Sappey—*i.e.*, a vein running alongside the round ligament of the liver, connecting the portal vein with branches of the epigastric veins near the navel. This often produces a large bunch of varicose veins, the so-called caput medusæ.

It will be thus seen that the later symptoms are dependent on a grave interference with the hepatic metabolism plus

general venous congestion. The toxæmia and exhaustion, consequent on serious obstruction, may bring about a fatal issue.

Biliary Cirrhosis.—The characteristic symptoms are—

Chronic jaundice, at first slight, but slowly becoming deep.

Absence of ascites.

Elevation of temperature, periodic in character.

Great enlargement of liver, which is firm, smooth, and painless.

Marked enlargement of spleen.

Early age of onset (20-30, and in children).

Chronicity of the disease (may last several years).

Prognosis.—Bad as regards cure; but if the collateral circulation be well established, the atrophy not marked, and the patient made to live temperately, bad symptoms may not arise for a considerable period. In far-advanced cirrhosis the treatment is merely palliative.

Treatment.—It must be (1) general, and (2) symptomatic.

General.—Absolute temperance in eating, abstinence from alcoholic drinks, and from hot condiments, etc., must be observed: the portal circulation should be depleted by the administration of a saturated solution of Epsom salts. Exercise, massage to the liver, and careful avoidance of chills, are the main points to remember. Ammon. chlor., arsenic, strychnine, etc., may be given.

Symptomatic.—In *ascites*, the fluid should be evacuated quickly by tapping, or by the slow method by means of Southey's tubes. However it is removed, the abdomen must afterwards be firmly bandaged, to avoid the consequences of too sudden relief of the pressure on the intra-abdominal veins. In lesser degrees of ascites, hydragogue cathartics are sometimes of use, but should be used with caution, as they may set up intractable diarrhœa. The hæmatemesis, if moderate, should not be treated too energetically, as it often acts as a safety valve by relieving portal congestion. If it be excessive, ice, recumbent position, ergot, acid sulph. dil., full dose of morphia, hydragris, hazeline, etc., should be resorted to.

The Emaciation calls for tonics, and the most nutritious diet compatible with the imperfect assimilative powers. Some authorities advise skim-milk diet.

Operative measures are sometimes employed to relieve a rapidly increasing ascites, where the collateral circulation has not been fully established. The operation is known as epiplo-
pexy. The abdomen is opened, the fluid is evacuated, and the omentum is then stitched to the parietal wall and included in the sutures closing the incision. By this means an additional collateral circulation is obtained. The operation should not be performed until repeated tappings have been tried in vain. The latest statistics show 50 per cent of failures, including 33 per cent of deaths.

CANCER OF THE LIVER.

Cancer of the liver may be primary, but is more often secondary to cancer elsewhere, especially in the gastro-intestinal tract, and in women in the breast or pelvic organs.

Ætiology.—Men are more frequently attacked than women : from forty to sixty years seems the most favourable period. The predisposing causes are similar to those of cancer elsewhere, viz., heredity, injuries, etc.

Pathology.—All types of cancer have been found in the liver, but the primary forms are nearly always epitheliomatous, and of the cylinder-celled variety.

Primary Cancer.—Two distinct types are distinguished—

1. The massive cancer, greyish white in colour, occupies a large portion of the liver, and is abruptly defined from the liver tissue.
2. Nodular cancer, in which the nodules vary much in size, and are irregularly scattered *throughout* the organ.

Secondary Cancer.—The organ becomes *enormously* enlarged, and the nodules can easily be felt through the emaciated abdominal walls ; indeed, they may be near enough to the surface

to be visible (the so-called "Farre's tubercles"). On section we find—

1. Extensive areas of fibrosis.
2. Extensive areas of fatty degeneration.
3. Extravasations of blood.
4. Dense greyish hyaline masses.

Symptoms of Cancer of the Liver.—Daily increasing emaciation, *hepatic pain*, malignant cachexia, jaundice, *sometimes* ascites, attacks of local peritonitis, together with the characteristic physical signs of the rough, uneven, enlarged liver, are the principal features. The nodules often present a characteristic central depression (umbilication). The organ is markedly *tender* upon pressure. It should, however, be remembered that cancer may be added to a cirrhosis of the liver. When the growth is of a melanotic sarcoma type, the pigmentation, plus other symptoms, may point to the true nature of the disease.

Diagnosis between cancer and cirrhosis is often difficult when there is a strong history of alcoholism. The following table may help—

	CIRRHOSIS.	CANCER.
<i>Progress</i> . . .	1. Often slow.	1. Always rapid.
<i>Liver</i> . . .	2. Enlarged at first, then smaller, and more nodular as atrophy becomes more marked.	2. Is large, and the nodular character developed from the first.
<i>Pain</i> . . .	3. Not marked.	3. Well marked.
<i>Ascites</i> . . .	4. Usually present.	4. Often absent.
<i>Jaundice</i> . . .	5. Not till late.	5. Often a marked feature.

HYDATID DISEASE.

Hydatid Cysts occur more frequently in the liver than in any other organ (see *Tænia*). They are usually single, but may be multiple. They are most frequent in the right lobe.

Symptoms.—A hydatid tumour of the liver grows slowly, and usually in one particular direction. It is painless (until it suppurates), and may cause no symptoms until it becomes extremely large or bursts.

Physical Examination.—Tumour is globular, tense, elastic, and with care fluctuation is detected. On palpation a peculiar vibratory thrill may be felt. There is no tenderness, nor any sign of ‘pointing,’ as in abscess. If the tumour is very large, there may be pressure upon the lung, the abdominal viscera, the vena cava, or the portal vein, causing respectively dyspnœa, constipation, œdema, or ascites.

Prognosis.—The cyst may undergo spontaneous cure without rupture. Death of the parasite causes the cyst to contract. Rupture may take place into the lung or pleura, into the intestines, into the hepatic duct, or into the peritoneal cavity.

Treatment.—As soon as the cyst is discovered, employ surgical means, such as—

1. Aspiration.
2. Careful incision, and free evacuation of cyst contents, after which the cyst wall is stitched to the abdominal wall, and a drainage tube is inserted. This is much the best method of treatment.

Other cysts of the liver may occasionally occur, as may *cavernous tumours*. Their interest is chiefly pathological.

WAXY LIVER.

The liver is often affected with lardaceous disease, and as elsewhere, the degeneration begins in the middle coats of the blood-vessels. The lobules are invaded from without inwards, the hepatic cells become destroyed, and the glandular structure converted into a firm, dense material. The liver is enormously enlarged, and smooth. On section, the substance is glistening, firm, and resembles yellow wax, the cut surfaces showing only faint traces of lobules. It must be remembered that waxy disease may co-exist with fatty liver, syphilitic disease, etc.

Causes.—Prolonged suppuration, as in strumous ulcerations, caries, necrosis, and phthisis; syphilis, even where there has been no suppuration; very rarely as a result of malignant fevers or the malarial cachexia.

Symptoms.—Sense of weight in the hepatic region, anorexia and other dyspeptic symptoms; diarrhoea, the stools being pale and often fatty. The liver is enormously enlarged, firm, smooth on the surface, and not tender. The spleen is also much enlarged, and presents similar characters. Later, there may be jaundice (very uncommon), ascites, and albuminuria, indicating implication of the kidney in the amyloid change. *Pain is rare.* The disease is usually fatal.

Treatment.—Remove cause as far as possible. Iodide of potassium is often of great service, especially when the disease is due to syphilis. The general treatment is the same as that of tuberculosis.

FATTY LIVER.

By fatty liver is meant a great increase in the quantity of fatty globules, naturally contained in the hepatic cells, so that on examination the cells are found loaded with oil globules, often obscuring the nuclei. The change takes place first at the periphery, but later the whole of the lobule is invaded. The liver is usually somewhat enlarged, with a smoothish surface.

Causes.—General obesity; emaciating diseases, such as cancer and phthisis; venous congestion may cause both nutmeg and fatty liver. Chronic alcoholism is an important cause, and fatty liver may be present from this cause with no vestige of cirrhosis. Care should be taken not to confound fatty infiltration with fatty degeneration. In the latter condition the hepatic cells undergo a complete metamorphosis. Phosphorus poisoning causes extreme fatty degeneration. Physiological chemistry leads us to anticipate fatty *infiltration* in those cases where the oxidising power is insufficient to dispose of the ordinary *fat-forming* elements of the daily food.

Symptoms.—Often are very obscure, but the symptoms of chronic gastric catarrh, constipation alternating with diarrhœa, pasty complexion, together with the physical signs of an enlarged liver, somewhat soft in consistence, regular in outline, and *not* accompanied by pain or jaundice, or by enlargement of the spleen, render the diagnosis fairly easy.

Treatment.—Regulate the diet, avoid excess of proteids or carbohydrates particularly. Prescribe alkaline aperients, chloride of ammonia, calomel and podophyllin, massage and exercise.

DISEASES OF THE GALL BLADDER.

Inflammation of the gall bladder is most frequently due to extension of inflammation from neighbouring parts, but it may be caused by direct irritation of the cystic mucous membrane through the passage or impaction of gall-stones, parasites, etc.

Dropsy.—When the *cystic* duct is blocked, the mucous membrane of the gall bladder undergoes a peculiar degeneration, resulting in a secretion of watery fluid, which may distend the gall bladder enormously. *Often the tumour is so freely movable as to cause it to be mistaken for a movable kidney.*

Gall-Stones.—Biliary calculi are most common in females between the ages of thirty and forty-five.

Composition.—

Single gall-stones are almost entirely composed of cholesterine (a monatomic alcohol). The nucleus is usually a bit of dried mucus, the result of a previous catarrh of the gall bladder. The stone is semi-translucent, glistening, with a somewhat *granular surface*, and is very light.

Multiple gall-stones are much more common; a few or a hundred stones may be present. They differ from the single stone by—

1. Being stratified: the strata are of different shades of colour, some being very deeply coloured.
2. Presenting facets on their surface from pressure.

Effects of Gall-Stones are—

1. To cause more or less inflammation of the mucous membrane.
2. Impaction in the ducts, with consequent jaundice, etc.
3. They may cause serious ulceration.
4. During their passage along the ducts they cause the intensely painful "biliary colic," and often
5. Subsequent appendicitis, obstruction of bowels, etc.

Symptoms of Biliary Colic—

1. Sudden and excruciating pain in the hepatic region, radiating all over the *thorax* to the right shoulder, and with a sense of general thoracic constriction. *Marked tenderness in epigastrium and right hypochondrium.*

2. Rigors, profuse sweating, great feebleness of pulse, symptoms of collapse.

3. Vomiting, which often gives relief (probably by the contractions of the diaphragm so induced aiding peristalsis).

4. *Subsequently* to the attack, jaundice is more or less pronounced, and there are marked prostration and slight fever during the next few days.

It must be remembered that (as in renal calculus) the stone, after obstructing for a time, may slip back again into the cystic duct or gall bladder, giving a sequence of events which may be several times repeated. Moreover, gall-stones may be the cause of hypertrophic cirrhosis, or the exciting cause of cancer. (Dr. SAUNDBY.) The stone may be impacted in the cystic duct, or in the common duct, causing permanent jaundice of varying intensity.

Many observers have shown conclusively that the presence of gall-stones may give rise to a "bastard ague."

Treatment of Biliary Colic—

1. Hypodermic injection of morphia.
2. Chloroform inhalations until morphia acts.
3. Stimulants to meet collapse.

Subsequently : purgatives, careful dieting, regular exercise, and avoidance of any constriction of the hepatic circulation by tight corsets, etc.

Persistent Biliary Obstruction.—Demands surgical interference, but care must be taken to diagnose the cause of obstruction first.

Cancer of the Gall Bladder arises usually near the neck. It is of the epitheliomatous type, originating in the mucous membrane. It causes rapid obstruction of the ducts.

Symptoms.—Intense and persistent jaundice, pain, often severe, anorexia, emaciation. Physical signs,—enlargement of liver, tumour projecting from its lower edge, pear-shaped, hard to the touch, and intensely tender.

Diagnosis of Gall Bladder Affections.—A *distended* gall bladder forms a *smooth ovoidal* tumour below the ninth right costal cartilage. Take into consideration the history, jaundice, character of pain, etc.

DISEASES OF THE PANCREAS.

These are comparatively rare, but not the less important. In a series of 6000 autopsies performed at Guy's Hospital, the pancreas was diseased in 99 instances, or 1·6 per cent (Hale White). Pancreatic diseases are important owing to their frequent association with diabetes (which occurs oftenest in connection with atrophy of the organ), and also from the definite symptoms which they cause.

Pancreatitis may be acute or chronic. In either case it is the result of infection extending along the duct from the intestine. The *acute* form may be either suppurative or hæmorrhagic, and may lead to thrombosis of the splenic vein and fat necrosis of the subperitoneal fat. It occurs in males between the ages of 25 and 60. The onset is sudden, but there may be previous duodenal catarrh. Severe epigastric pain, irregular vomiting, meteorism, fever, shiverings, and often collapse, are the chief symptoms. Slight jaundice may

occur. The disease is usually fatal. *Treatment* is mainly dietetic. Relieve pain by morphia.

The *chronic* form has no very definite symptoms. It leads to generalised fibrosis with atrophy of the glandular substance (diabetes). Cysts or calculi may form in the ducts.

Pancreatic Cysts, if small and numerous, may be due to chronic pancreatitis; if large, there may be no inflammatory change. They may reach a great size. The fluid contents possess diastasic activity, an important point in diagnosis. The smaller cysts cause no symptoms; the larger cause tumour in the epigastrium (often with visible bulging) median in position, immobile, elastic but non-fluctuant, and dull to percussion. Pain, fever, and wasting are absent. Inflation of the stomach shows that the tumour lies behind it. The *treatment* is surgical.

Cancer of the Pancreas affects chiefly the head of the gland. It may be scirrhus or encephaloid, and causes metastatic growths in the liver and spleen. It may press upon the duodenum (dilatation of stomach) or infiltrate the hilus of the liver (jaundice, ascites). The tumour often reaches a considerable size. The early *symptoms* are severe and paroxysmal epigastric pain, with rapid wasting, and the other general signs of malignant disease. Later a tumour, or sometimes only increased resistance, is to be felt in the upper part of the abdomen, to the right of the middle line. The tumour, when present, is firm, tender and immobile. Pressure symptoms (see above) may be noted. Glycosuria is sometimes present. Death occurs from asthenia or gastric hæmorrhage.

DISEASES OF THE PERITONEUM.

ASCITES.

ASCITES or dropsy of the peritoneum is not a disease in itself, but is merely symptomatic of some condition which causes an increased transudation of fluid into the peritoneal cavity. Dropsy means some abnormal change in the blood-vessel walls, either the result of inflammation, vasomotor paralysis, or obstruction; and since we know that the venous blood of the intestines is returned *via* the portal circulation, we naturally look for the more common causes of ascites in morbid states of the portal vein; but it must be remembered that inflammatory changes of local blood-vessels in connection with morbid processes set up in the peritoneum and various abdominal organs tend to bring about dropsy of the peritoneum. It is highly important to remember that the causes of ascites are *not confined to obstruction* of the portal vein; sometimes the dropsy is of local inflammatory origin, and the fluid is then frequently collected into localised pools—*i.e.*, within the mesh-work of inflammatory adhesions. The typical signs of ascites are often masked by the large deposits of inflammatory lymph between the intestines, etc.

Causes.—The more common causes of ascites tabulated are—

1. *Portal obstruction*, either within or outside the liver.
2. Phlebitis of the portal vein.
3. Disease (usually malignant or tubercular) of the peritoneum itself.
4. Morbid states of the blood associated with renal disease, leucocythæmia, etc.

It must be remembered that obstruction of a vein does not of itself produce dropsy *until some retrograde change is set up in the walls of the vessels*. Obstruction must of course produce such changes in time.

Under portal obstruction we may mention cirrhosis of the liver, neoplasms of the liver, pancreas, spleen, etc. ; malignant disease of, or growths in connection with the intestines, and other abdominal organs.

When ascites is caused by cardiac or pulmonary disease, probably it is due to two factors, viz., portal congestion, and a *deteriorated character of the blood*.

Diagnosis.—The cardinal symptoms are—

1. Enlargement of the abdomen, most marked in the flanks when the patient is lying on his back : the skin is tense and shiny with dilated superficial veins on the surface.

2. Prominence of the umbilicus.

3. Fluctuation, and vibration on palpation.

4. Percussion yields a dull note, which alters on the patient assuming a new position of the body.

5. The above conditions may be associated with anasarca, or other symptoms of dropsy elsewhere.

When the patient lies on the back, the dulness on percussion is most marked in *the flanks and hypogastrium* ; whilst the note is *resonant* at the umbilicus. Turn the patient on the side, the fluid moves to the most dependent part, and consequently a resonant note may be obtained in the *opposite* flank.

Caution, however, is necessary as regards this resonance ; it may be absent through adhesions binding the intestines down to the posterior wall ; or the fluid may not gravitate, *because of being hemmed in by inflammatory lymph*. On the other hand, if the fluid be small in amount and the distension of the intestines marked, the *dulness* may be difficult to demonstrate.

Treatment.—Seek the primary disease, and direct the treatment to improving the *general* health of the patient on the principles already laid down under hepatic disease.

If the fluid causes much dyspnœa, or other symptoms, it should be evacuated by Southey's tubes, or by tapping. Paracentesis may be repeated many times without danger if due antiseptic precautions are observed. It must not be forgotten that in old cases the vessels of the abdomen may be so far devitalised, that a sudden removal of the support afforded them by the dropsical fluid causes them to become engorged with blood, and may set up fatal syncope. The abdominal wall should therefore be supported during the operation by bandages, gradually tightened as the fluid is withdrawn.

DIAGNOSTIC TABLE.

OVARIAN TUMOUR.	PREGNANCY.	ASCITES.
1. Growth begins on <i>one</i> side. Dulness does not change with position of patient.	1. Begins in median line.	1. Fluid moves on altering position. Swelling is uniform.
2. Slow growth.	2. Uniform and definite rate.	2. May be rapid or not.
3. Fluctuation general over the tumour.	3. Fluctuation absent except in hydramnios.	3. Fluctuation general over the whole abdomen.
4. No signs of pregnancy, and health deteriorated.	4. Signs of pregnancy, and health normal.	4. No signs of pregnancy, and health much impaired.

In all cases an examination should be made of the ascitic fluid, both microscopically and macroscopically.

Ordinary Ascitic Fluid.—Is light yellow or straw coloured, generally of about 1010 sp. gr. ; contains albumin. In *chylous* ascites associated with disease of the pancreas and lacteals the fluid is turbid and milky, exhibiting oil globules. In malignant ascites, the fluid is often dark from the presence of blood. "Cancer" cells may be discovered under the microscope.

Fluid due to inflammatory exudation is of a higher sp. gr., 1014-1020.

PERITONITIS.

Inflammation of the peritoneum may be either of an acute or of a chronic nature. Recent and extensive observations

in the mortuary go to show that primary or idiopathic peritonitis is seldom or never seen. In order to understand this, I cannot do better than borrow Dr. Fagge's words: "The peritoneum is a huge areolar space or lymph sac, and its most intimate pathological relations are *not* with skin or mucous membrane, *not* even with joints, or the so-called arachnoid space, but with pleura, pericardium, and tunica vaginalis; all of which parts are embryologically parts of the great body cavity, formed by the splitting of the mesoblast into somatopleuræ and splanchnopleuræ. The diseases of these three divisions of the same original cavity are the same: *acute* inflammation, serous or purulent, traumatic or septic, chronic irritative effusion and passive dropsical effusion, hydrothorax, hydropericardium, and ascites; chronic adhesive inflammation with hypertrophy. All three are liable to be invaded by tubercle and also by cancer. All three are prone to follow the pathological fate of the viscera which they cover; they are all apt to suffer in the course of Bright's disease, and, lastly, they are often all affected together by inflammation, by tubercle; or more rarely by cancer." (Second edition of Fagge's *Medicine*.)

A peritonitic effusion differs, however, from pleuritic effusion in showing a strong tendency to become purulent. Possibly this purulent state is to be explained by the close proximity of the septic intestinal contents. Peritonitis is usually due to bacterial infection, either by the pus cocci or intestinal organisms.

Varieties.—1. Acute—divided into

(a) Local	} peritonitis.
(b) General	

2. Subacute—in which form attacks are nearly always due to the action of local irritation.

3. *Chronic*.—This form is usually due to irritation from specific, infective, or malignant growths—*e.g.*, chronic dysentery, tuberculosis, syphilis, cancer, etc.

Symptoms.—The symptoms of a general acute peritonitis present a most characteristic clinical picture, the main features of which are—

1. Great pain and tenderness over the abdomen, which is usually tense from tympanites.

2. Quick, wiry, and incompressible pulse.

3. *Facies Hippocratica*—*i.e.*, “a sharp nose, hollow eyes, collapsed temples; the ears cold, contracted, and their lobes turned out; the skin about the forehead being rough, distended and parched; the colour of the whole face being brown, black, livid, or lead colour.” Face is also anxious.

4. Constipation.

5. Vomiting and often hiccough.

6. Dry, small red tongue.

7. Moderate fever.

8. Moderate and uniform abdominal distension, increased resistance, *absence of visible peristalsis*. In later stages dulness in flanks (fluid effusion).

9. In perforative peritonitis the abdomen may be tympanitic all over, the hepatic and splenic dulness being obliterated.

The abdominal tenderness is sometimes so marked that the slightest touch or even the respiratory movements—sneezing, passing flatus, and coughing—causes exquisite agony. Possibly the constipation, and the small wiry pulse, may be due to paralysis of the splanchnic nerves. Whilst the above are the cardinal symptoms of a general acute peritonitis, it must not be forgotten that the temperature may be sub-normal, diarrhoea be present instead of constipation, and the patient's face may be actually *apathetic* instead of anxious. This latter state is probably more frequent when the peritonitic fluid speedily becomes purulent, as seen in some cases of puerperal fever, typhoid fever, and strangulated hernia.

Causation.—Nearly always secondary to perforations of some viscus, gastric ulcer, typhoid ulcer, obstructed bowel, etc.; or to extension of inflammation of neighbouring parts, such as ovaritis, salpingitis, parametritis, etc.; or to blood

poisons, such as malignant fevers, especially puerperal fever; or to morbid state of the blood, as seen in Bright's disease, etc.

Pathology.—

1. Hyperæmia with loss of lustre; the hyperæmia is most marked where the intestinal coils *are not* in close contact with one another.

2. Exudation of lymph, giving a more or less shaggy appearance.

3. Effusion of fluid, which may be highly fibrinous and coagulate easily, forming extensive adhesions; or which may become

4. Rapidly purulent.

Prognosis.—Depends largely on the cause. When the disease is the result of perforation and extravasation of blood into the peritoneal cavity, it may be fatal in a few hours. The prognosis is of course much more favourable when the peritonitis is localised.

Treatment.—Almost entirely operative. The extent of the operation depends upon the local conditions found when the abdomen is opened, and upon the general condition of the patient. Operation should be done as soon as possible; if for any reason it is delayed, the previous treatment must consist mainly in alleviating pain and lessening peristalsis by opium or morphine, and locally by ice or warmth, whichever is best borne. It is desirable not to mask the symptoms by using morphine too freely, but that is not to say that the appeal of humanity should be neglected. Where there is much shock or collapse, intravenous injection, or injection into the subcutaneous cellular tissue, of normal saline solution is of great value. Large quantities may be used. Besides producing a temporary restoration of the strength such as may fit the patient for operation, these injections tend to eliminate the toxins by flushing the kidneys.

In peritonitis due to perforation, the prospect of recovery is in inverse ratio to the amount of delay in operating.

Chronic Peritonitis includes at least three well-marked types, *i.e.*—

1. Peritonitis, as a result of the invasion and subsequent breaking down of tubercles.

2. A form associated with, and due to irritation of malignant growths.

3. A localised form associated with chronic inflammation, diseases of certain organs—*i.e.*, the uterus and appendages—chronic constipation, and other affections of the lumen of the intestines.

Symptoms vary with the cause, the extent of the effusion, and adhesions.

Constipation alternating with diarrhœa, colicky pains, loss of flesh, exacerbations of fever, and localised tenderness, are the chief symptoms complained of.

Though ascites is often prominent, much more frequently the fluid is collected into small pools in the meshes of the bands of lymph.

The mesentery is usually much shortened, and the calibre of the gut diminished,

Physical Examination.—May reveal hard masses of tubercular or malignant growths, etc. The abdomen may be either flat and dull, or distended and resonant.

Treatment.—First seek the cause, and, if possible, remove it. In the early stages of chronic tuberculous peritonitis the diet should be highly nutritive, and especially rich in fats. The bedroom should be freely ventilated, and whenever possible the patient should be much in the open air. Mercurial inunction is sometimes useful. Cod-liver oil, malt, etc., and iodoform given internally, are often beneficial. Massage, with the administration of the iodides and tonics, may be of use. Laparotomy is often successful in tuberculous peritonitis. The precise reason for this is not known. It must not be forgotten that chronic peritonitis is often fatal through an acute exacerbation of inflammation.

TABES MESENTERICA

Is a disease due to tubercular degeneration of the mesenteric glands. Though associated with tubercular peritonitis it may be a primary disease, especially in infants and young children.

Pathology.—The glands are much increased in size, and all stages of tuberculosis may be seen; in some cases the glands are soft from fatty or caseous degeneration; in others, again, they show a great increase of the fibrous tissue elements, and may consequently be harder than normal.

Symptoms.—A condition which so seriously interferes with the lacteal absorption must of necessity cause grave symptoms of malnutrition; and, indeed, the cardinal symptoms of tabes mesenterica may be summed up—as great wasting of the limbs, thorax, and face, with a *large* abdomen, unhealthy action of the bowels, and general exhaustion. The abdomen may be (as pointed out in Chronic Peritonitis) either resonant or dull. The enlarged glands can be distinctly felt, and ascitic fluid is often present. Diarrhœa usually alternates with constipation. The stools are slimy and pasty in character; they often contain undigested milk, fat globules, and sometimes much brown pigment.

Diagnosis.—Care must be taken not to diagnose as tabes mesenterica, those cases of malnutrition with large abdomen, the result of improper feeding, congenital syphilis, and rickets. A careful examination of the abdomen should prevent such mistakes.

Treatment.—Everything must be done to put the child under the most favourable hygienic conditions, as detailed under Rickets. Iodide of iron is often most useful. Iodoform applied at night to the abdomen, in the form of an ointment spread on strips of calico, followed by gentle massage in the morning, sometimes does a great deal of good.

DISEASES OF THE CIRCULATORY SYSTEM.

[THE matter printed in small print is taken by permission from Dr. Wyllie's original notes and diagrams.—Though printed in smaller type the reader must not think it less important on that account ; on the contrary, I think it a most essential part of the book.—A. WHEELER.]

PHYSICAL EXAMINATION.

PERCUSSION.

1. In health the area of *Superficial Cardiac Dulness* is of triangular shape ; the apex or upper angle (truncated) reaches as high as the fourth left costal cartilage ; the right border descends vertically along the left border of the sternum ; the left border passes obliquely downwards and to the left until it reaches the outer limit of the apex beat ; the base cannot be percussed out owing to the proximity of the Liver, but corresponds to a line drawn from the outer and inferior limit of the apex beat inwards until it meets the perpendicular limit of cardiac dulness, about mid-sternum. The measurement of the basic line is important ; normally it measures about three or four inches.

2. The area of *Deep Dulness*, obtained by heavy percussion, corresponds in shape to the area of superficial dulness, but is more extensive. It overlaps it about an inch on every side.

NOTE.—In Disease the areas of Superficial and Deep Dulness may be specially extended to the right or left, according as the right or left chambers of the heart are specially enlarged.

3. The percussion of the region of the *Aortic Arch* (above the level of the third costal cartilages) is especially important in cases of aneurism and of mediastinal tumour.

PALPATION.

1. Of the *Precordia*.

- (a) Determine the *position* of the apex beat. The normal position is between the fifth and sixth ribs, about half an inch within the vertical line of the nipple. When the left ventricle is enlarged there is displacement of the apex beat downwards and to the left. When the right ventricle is enlarged there is *apparent* displacement of the apex beat to the right, and there is often pulsation in the epigastrium.
- (b) Note the *limitation* or *diffusion* of the apex beat.
- (c) Note the force and character of the beat: whether moderate and deliberate, as in health; or strong and sudden, as in nervous excitement; or strong and slow (heaving) as in hypertrophy; or weak or imperceptible, as in debility.

The range of variation is considerable even in health, owing to the shape of the chest, etc.

2. Of the *Aortic Region*. In cases of suspected aneurism note presence or absence of pulsation.

3. Of the *great vessels at the root of the neck*. Venous pulsation is scarcely palpable, though strikingly visible; arterial pulsation is as strikingly palpable as visible.

INSPECTION.

1. Of the *Precordia* and *Aortic Region*.

- (a) *Form*. Is there bulging over the precordia or over the aortic region?
- (b) *Movements*. (1) Movement of the apex beat; its situation, amount, and diffusion; (2) Pulsation in the Epigastrium; (3) Pulsation in the region of the Pulmonary Artery, common in anæmic debility; (4) Pulsation in the Aortic Region, often present in cases of aortic aneurism.

2. Of the *great vessels of the neck*. (a) Fulness of the great veins; (b) Pulsation in these veins; (c) Excessive pulsation in the arteries.

3. Of the *General Circulation*: as exhibited in the patient's complexion, the condition of his peripheral arteries and veins, the presence or absence of dropsy, etc.

4. Of the *Pupils*, in cases of Aneurism of the Aortic Arch.

EXAMINATION OF THE RADIAL PULSE.

1. Give the *Pulse Rate* per minute.

2. Give the *Rhythm*: regular or irregular; if intermittent, note the average proportion of the intermissions to the pulse-beats.

3. Note the size, force, and character of the *Blood-wave*: large, moderate, or small; deliberate or sudden: strong or weak; a double wave (dirotism).

NOTE.—In *weak heart* the radial pulse may be almost or wholly imperceptible ; or only a proportion of the heart's contractions may produce blood-waves sufficiently strong to be propagated perceptibly to the radial artery, and thus the radial pulse may appear to be much slower than the rate of the heart's contractions ; or the weak pulse may be affected by the patient's respiration, its beat being weakened by Inspiration and strengthened by Expiration.

4. Between the beats of the pulse, test particularly the *Resistance* of the artery to *pressure*. Marked resistance may be due either to rigidity of the artery's coats or to high blood-pressure. Press the artery firmly against the bone, and examine the coats by rolling it beneath the finger.

5. Compare the two radial pulses, and note any *difference in strength*, or *want of synchronism*, in their beats.

6. In special cases take a sphygmographic tracing.

AUSCULTATION.

ENDOCARDIAL MURMURS OR BRUITS.

1st Sound marks beginning of Systole. Systole continues through nearly whole of short pause.

2nd Sound marks beginning of Diastole. Diastole continues through nearly whole of long pause.

1. AORTIC—



Systolic.
(Obstructive.)



Diastolic.
(Regurgitant.)



Systolic and Diastolic
(Double.)

2. MITRAL—



Systolic.
(Regurgitant.)



Presystolic.
(Obstructive.)



Presystolic and Systolic.
(Double.)

1. AORTIC MURMURS. As shown in the above diagram, there may be at the Aortic Orifice, a Systolic murmur indicative of obstruction, or a Diastolic indicative of regurgitation ; and these murmurs are frequently combined so as to constitute a Double Aortic murmur. A Systolic murmur is not always due to organic disease, being sometimes of Anæmic origin, as will be explained below, under "6. The Murmurs of Debility." This Hæmic murmur is usually softer than a murmur of Organic origin. Aortic murmurs, organic and hæmic, are produced at the Aortic Orifice, which is situated at the Sternal Articulation of the third left costal cartilage. They are thus Basic murmurs.

2. MITRAL MURMURS.—These are heard best at the apex of the heart. A Systolic murmur indicates Regurgitation. This may be due either to Organic disease of the Valve, or to dilatation of the Ventricle and its Auriculo-ventricular Orifice, the latter causing a “murmur of debility,” owing to “Disparity of size” between the dilated orifice and its valve (“relative insufficiency”). The Obstructive murmurs are almost always Organic, being due to stenosis of the Mitral valve. Two forces cause the blood to flow through the Mitral Orifice during the diastole of the Ventricle, viz., the suction of the Ventricle (*a vis a fronte*), and the propelling force of the Auricle (*a vis a tergo*). The suction is strongest near the beginning of the diastole, and the propelling force at the end of it, immediately before the Ventricular Systole. When there is obstructive disease, the murmur is developed at the time when the flow of blood through the contracted orifice is rapid enough to produce a murmur. Generally it is limited to the period of Auricular contraction, and is therefore Presystolic. Sometimes, when the Auricle is weak, it occurs only at the period of greatest Ventricular suction, and is therefore Diastolic. Occasionally, again, it may be both Diastolic and Presystolic. Obstructive murmurs are rough and purring. They are often succeeded by the blowing murmur of Regurgitation, since the disease which produces obstruction often renders the valve incompetent at the same time. A common double Mitral murmur is thus a rough, obstructive Presystolic, running up to, and immediately succeeded by, a blowing, Systolic, regurgitant murmur. The relations of these murmurs to each other are shown in the diagrams. In sound the rough Presystolic murmur might be represented by the letters *rrrp*, the terminal *p* representing the first sound of the heart, which, in such cases, is often loudly accentuated. A double Mitral murmur may be represented by the letters *rrffff*.

3. MURMURS AT THE PULMONARY ORIFICE correspond in time to Aortic Murmurs. A Systolic murmur in this situation is pretty common. It may be “functional,” and due to Hæmic causes, as Anæmia; or it may be due to organic disease that has produced Pulmonary stenosis—a form of congenital heart disease. A Diastolic murmur at this orifice is extremely rare.

4. TRICUSPID MURMURS correspond in time to Mitral. Practically the only Tricuspid murmur that is not extremely rare is a Systolic Regurgitant. It is a “murmur of disparity of size,” and is due to enlargement of the orifice without corresponding enlargement of the cusps, a condition that is always present when there is much dilatation of the ventricular chamber. With this murmur venous pulsation in the neck is generally associated.

5. In cases of ANEURISM a Systolic murmur over the sac is pretty common. In very rare cases a double murmur (Systolic or Diastolic) is produced by the flow of blood into and then out of the sac. There may be a double murmur in aneurism of the first part of the arch, where aortic regurgitation co-exists with the aneurism. In many cases there is no murmur at all.

6. The MURMURS OF DEBILITY, so common in Anæmia, are both Vascular and Cardiac.

The *Vascular* murmurs are (*a*) The *Arterial* murmur, systolic in time, heard over the great arteries of the neck. This is generally supposed to be common, but it is very often produced artificially by the pressure of the stethoscope. (*b*) The venous hum (the humming-top murmur, or "Bruit de Diable"), heard over the great veins of the neck, and sometimes over other large veins, such as the ophthalmic veins and the cerebral sinuses. This is very common and important.

The *Cardiac* murmurs of Debility are variously classified and explained. A view largely supported is that they are *four* in number, one for each orifice of the ventricles: that two are therefore basic and are heard over the Aortic and Pulmonary regions respectively, being probably due to the onward rushing of the thin anæmic blood through the Aortic and Pulmonary orifices; and that the other two, Mitral and Tricuspid, are heard over the right and left apices, and are due to dilatation of the ventricular chambers, which has produced the "disparity of size" between the auriculo-ventricular orifices and their cusps, already alluded to. Both of the latter are thus regurgitant.

Of these *six* murmurs (Vascular and Cardiac) *three* are common in Anæmia, namely, (1) the Bruit de Diable in the neck, (2) the Basic murmur in the Pulmonary region, (3) the Mitral murmur at the apex; and that is the order of their development.

Observe that all the Cardiac murmurs of debility are *Systolic* in time. Systolic murmurs may thus be either of functional or of organic origin, while Presystolic and Diastolic murmurs are always of organic origin.

7. EXOCARDIAL MURMURS. (*a*) *Pericardial friction*, due to Pericarditis, is generally a "to-and-fro" or double murmur (Systolic and Diastolic). It is most apt to be confounded with a double Aortic murmur, but its superficial rubbing and shuffling character generally renders the distinction easy. In most cases it appears first at the base of the heart, and spreads thence, if not arrested, over the whole organ. (*b*) A to-and-fro friction sound, of precisely the same character as the above, is sometimes produced by a *Pleurisy in the Precordial region*, the subjacent heart causing the inflamed surfaces of the pleura to rub against each other synchronously with its own movements.

PROPAGATION OF ENDOCARDIAL MURMURS.

1. AORTIC MURMURS are clearly heard about the third left costal cartilage at its junction with the sternum, that being the position of the Aortic valve. They are propagated to a distance by *three* agents, namely, (*a*) the *Heart* itself, which often carries them to the Apex; (*b*) the *Aorta* and its great branches, a spot of special importance in this respect being the junction of the second right costal cartilage with the sternum (here the Aorta makes its first bend, and Aortic murmurs are often heard even more distinctly than over the

valve itself); and (c) the *Sternum*, which often conducts the sonorous vibrations of such murmurs throughout its whole length. Obstructive Aortic murmurs (Systolic) are carried best upwards, in the direction of the blood current, and are specially loud over the first bend of the aorta. Regurgitant Aortic murmurs, produced by a descending current, are carried best downwards; and are very often heard better at the left edge of the sternum, close above its junction with the xiphisternum, than even over the Aortic valve.

2. PULMONARY MURMURS, starting like the Aortic from opposite the third left costal cartilage at the Sternum, are carried obliquely upwards and to the left, in the second left costal interspace, for a distance of about two inches, the agent of propagation being the trunk of the Pulmonary artery.

3. MITRAL MURMURS are loudest at the apex. The Regurgitant (Systolic) is propagated upwards and outwards towards the axilla and the angle of the scapula. The Obstructive (Presystolic and Diastolic) are not propagated in any special direction.

4. TRICUSPID MURMURS are heard best over the right ventricle, being audible over an area of some inches in diameter, whose centre is situated at the left edge of the sternum, close to its junction with the xiphisternum.

PERICARDITIS.

Inflammation of the pericardium is never idiopathic. It may arise by direct extension of inflammation, from infective processes, or from constitutional diseases.

Exciting Causes.—

- (1) Rheumatic fever.
- (2) Eruptive fevers, especially *scarlet* fever.
- (3) Septicæmia.
- (4) Tuberculosis.
- (5) Gouty state of blood.
- (6) Bright's disease.
- (7) Extension of inflammation from neighbouring parts

The disease is more frequent in the male, and occurs chiefly in early adult life.

Pathology.—That of inflammation of a serous sac—*i.e.*,

1. Hyperæmia, with loss of lustre.
2. Exudation of lymph, which coagulates and gives a peculiar shaggy, or “bread and butter sandwich” appearance

(*cor villosum*). The process might stop at this stage, causing the dry or *plastic type of pericarditis*. Much oftener the process goes on to the next stage.

3. Effusion of fluid.

4. Absorption with more or less adhesions, which may or may not (as we shall see) subsequently hamper the heart permanently.

The fluid is serous or sero-fibrinous; it may, however, be purulent or hæmorrhagic when associated with tubercle or malignant disease.

Symptoms.—The symptoms are somewhat obscure, and may be masked by the previous existing disease. Taking a typical case as it occurs in the course of rheumatic fever, we usually get—

1. Precordial distress—sharp pain is rare, when present it is most marked at the lower end of sternum.

2. Moderate fever, or exacerbation of already existing fever, at onset.

3. *Dyspnœa, and dusky appearance* of the face.

4. Rapid action of the heart, sometimes with feeble pulse.

5. Symptoms due to pressure by the fluid on the neighbouring organs (trachea and œsophagus, etc.)

6. Great restlessness.

It will be easily understood from the above table that we must rely more upon physical signs than subjective symptoms for diagnostic information.

Physical Signs.—

1. *Before Effusion of Fluid.*—On auscultation is heard the characteristic “*to and fro*” *friction rub*. It usually begins at the base of the heart and then extends more or less over the whole surface. The friction may sometimes be felt by the hand (friction fremitus). As the effusion takes place the friction becomes less pronounced, but is *rarely entirely absent at the base* until complete resolution or organisation takes place.

2. *Effusion Stage*.—The physical signs are—

- (1) *Marked increase of the cardiac dulness.*
- (2) Displacement of the apex beat.
- (3) Muffling of the heart sounds.
- (4) Displacement of other organs (if effusion be great).

The shape of the dulness is characteristic. It is conical, *the apex of the cone being truncated, and situated at the level of the second rib, owing to the close attachment of the pericardium to the great vessels.* The apex beat is generally pushed upwards and to the left. It lies, when it is palpable at all, distinctly *within* the left border of cardiac dulness, not, as in enlargement due to valvular disease, in close relationship to it. The marked distension of the pericardial sac surrounds the heart with fluid, and causes a dulness extending much beyond the limits of the organ itself. The amount of bulging and displacement of organs will, of course, vary with the amount of fluid present. As resolution takes place the friction returns, and may be very coarse in character. Muffling of the heart sounds is not always present, and is not entirely due to the presence of fluid. The foetal heart is quite distinctly heard through an amount of fluid greater than is usually present in pericarditis. The muffling is due to weakness of the cardiac muscle from accompanying myocarditis. Of course, in serous and in chronic pericarditis, the quantity of fluid may be very great.

Diagnosis.—Often difficult in *fat* people suffering from acute rheumatism, and also when great dilatation of the heart is present. Remember, however, the “*conical*” dulness of the pericardial effusion. The friction rub may be simulated by friction of the pectoral muscle. It is by no means uncommon to detect friction sounds over “rheumatic” muscles. The “pleuritic” rub can be readily distinguished by the difference in time. Do not forget that both pleurisy and pericarditis may, however, co-exist. See “Exocardial Murmurs.”

Prognosis.—Depends on cause ; that of simple sero-fibrinous pericarditis is good, and the fluid may be absorbed in a com-

paratively short time. Permanent damage to the heart may result from complication with endocarditis or myocarditis. The greater the amount of fluid, the worse is the prognosis, which becomes very grave if the fluid is purulent or hæmorrhagic. Pericarditis complicating renal disease is often fatal.

Adherent Pericardium.—After absorption of the fluid, the sac may be almost or quite obliterated by adhesions. Symptoms of this condition may remain latent, but in other cases there may be signs due to permanent and extensive adhesions—

1. The heart may be hypertrophied.
2. A peculiar retraction in the “apex” region during *systole*.
3. Diastolic rebound of the chest wall.
4. Collapse of the cervical veins during diastole (Friedreich’s sign).
5. *Pulsus paradoxus*, the pulse becoming smaller at the end of inspiration.

None of these signs is by itself pathognomonic, but taken together they establish the diagnosis.

Treatment.—

1. *Absolute rest in bed.*
2. A fly blister over the præcordia.
3. An hypodermic injection of morphia if pain be severe.
4. Treat cause—if due to “rheumatic” poison be careful in the use of salicylates. They tend, if given early, to prevent cardiac lesions; but when these are established their depressant action in large doses may be harmful. Salicin is better in such cases.
5. If heart failure threatens, give digitalis, *strychnine*, and diffusible stimulants. Paracentesis may be considered if the effusion is very great, and does not tend to become less.

To promote absorption after the acute symptoms have subsided,—

1. Blister.
2. Purge with hydragogue cathartics—*e.g.*, pulv. jalapæ co.
3. Administer iodides or chloride of ammonium.
4. When these fail, paracentesis should be performed.
The puncture should be made one inch from the left sternal margin in the fourth or fifth interspace. If the fluid be purulent, incise and drain.

ENDOCARDITIS.

By endocarditis is meant inflammation of the lining membrane of the interior of the heart, the valves being most commonly affected. It may be acute or chronic. The *acute* form is divided into the simple and malignant or ulcerative forms.

Ætiology.—Endocarditis is rarely a primary disease, but like pericarditis, which we have already discussed, secondary to other affections, and again we must emphasise the close relationship (before referred to) that exists between endocarditis, acute rheumatism, and chorea.

Malignant endocarditis may be primary, but is much more frequently the result of some septic or profound morbid change in the blood, such as diphtheria, scarlet fever, pneumonia, etc. The malignant form is not nearly so often associated with rheumatism and chorea as the simple endocarditis.

The distinction between simple and malignant endocarditis, it should be noted, is by no means absolute. The same organisms may be found to exist in either form, and even clinically a case may commence as simple, and end as malignant, endocarditis, or *vice versa*. In many cases, however, the distinction is sufficiently marked to warrant a differentiation between the two types.

Pathology.—*Simple Form*—

1. Hyperæmia of the membrane of the valves.
2. Exudation of lymph and proliferation of cells.
3. Formation of small granulations.

4. Deposit of layers of fibrin from the blood, the whole process resulting in the formation of small vegetations. These vegetations are most marked at a slight distance from the free borders of the valves—*i.e.*, those parts which come into opposition during closure.

Malignant Form (Ulcerative Endocarditis).—The initial changes are similar, but there are some important differences, inasmuch as ulcerations may *completely replace the vegetations*. When tabulated, the pathological differences are—

1. The vegetations when present are larger.
2. They have suppurating bases.
3. They contain colonies of micrococci. Both forms of endocarditis are really of microbic origin, and organisms are to be found in the vegetations and the substance of the valves. In malignant endocarditis, however, we have to do with the *pyogenic* organisms, *staphylococcus pyogenes aureus* et *albus*, and *streptococcus pyogenes*.
4. When they become detached they form *septic* emboli, giving rise to metastatic abscesses.
5. The ulcerative process causes great destruction of the valves, and may even lead to perforation of the curtains.
6. The subsequent or permanent changes in the valves, if the patient survive, are much more marked.
7. If the vegetation touches the endocardium as it flaps to and fro, the part touched is inflamed from contact.

As regards the side of the heart most affected,

1. *Congenital* endocarditis attacks the right side of the heart.
2. The simple endocarditis attacks the left only.
3. The malignant attacks *both sides*, though the *left* is much more implicated than the right side.

The vegetations are upon that side of the valve opposed to the blood stream—*viz.*, at the aortic valve the vegetations project into the ventricle, at the mitral valve into the auricle.

Symptoms.—

Simple Endocarditis.—The signs are extremely ill marked ; possibly increased rapidity of pulse, dyspnœa, præcordial distress, etc., may attract attention to the heart. On examination a *recently developed murmur* of a soft blowing or bellows-like character may be heard in the mitral or aortic areas. The commonest murmurs are those of mitral regurgitation (systolic), or mitral stenosis (presystolic).

It should be remembered, however, that in most fevers the heart is somewhat dilated, and a murmur, *not due to endocarditis*, may be present. We must, therefore, be cautious in coming to a too rapid conclusion that a suddenly developed murmur is indicative of endocarditis. An important distinction is that the onset of endocarditis is usually accompanied by a smart rise in temperature above the previous level, while in hæmic murmurs, or those due to simple dilatation, this is absent. A diastolic murmur in the aortic area is likely to be organic (aortic regurgitation).

Malignant Form.—Two types are described.

1. *The Septic Type.*—Is characterised by the symptoms of septic infection—viz., rigors, sweats, oscillating temperature, emaciation and metastatic abscesses. Often this form occurs in patients afflicted with chronic valvular disease. The symptoms may continue for months.
2. *The Typhoid Type.*—Is characterised by irregular or intermittent temperature, looseness of bowels, petechial rashes, and a rapid assumption of the typhoid state. Great difficulty may be experienced in diagnosing this form from typhoid fever or meningitis.

Along with these general symptoms there are usually, in both types, definite cardiac signs,—development of murmur, dilatation of the heart, cardiac irregularity, and so on. But the cardiac symptoms may be altogether latent, causing difficulty in diagnosis.

In both simple and malignant forms vegetations may be detached from the affected valve, and plug the smaller arteries. In the simple form these emboli are of importance when they implicate end-arteries (brain, lungs, etc.), and they then result in hemiplegia, hæmorrhagic infarction of lungs, and so on. In the malignant form the same results are produced when end-arteries are affected, with subsequent abscess-formation, while in the case of other arteries metastatic abscesses ensue. Obviously the risk of detachment is greater in the ulcerative type.

It should not be forgotten that patients with chronic valvular disease may get frequent attacks of sub-acute endocarditis.

We must also remember that endocarditis is never a solitary process. To some extent the myocardium always shares in the inflammatory changes, and the prognosis, in cases of chronic endocarditis following upon the acute form, depends as much upon the condition of the cardiac musculature as upon the actual valvular lesion.

Treatment.—All forms of endocarditis require absolute rest. The primary disease should be treated, and special treatment directed to prevent heart failure. It is impossible to lay down any hard-and-fast treatment beyond repeating, *that the utmost vigilance should be kept up for signs of syncope or embolisms*, and such conditions treated on rational grounds. Much that has been said in regard to the treatment of pericarditis may be said of endocarditis.

CHRONIC ENDOCARDITIS.

Chronic Endocarditis is usually the result of an acute endocarditis, but may be sub-acute in its onset, as a result of alcoholism, syphilis, gout, and Bright's disease. Amongst other factors is vascular strain, however brought about. One or more valves may be affected.

Pathology.—In those cases not directly attributable to acute endocarditis, the changes briefly are—

1. Formation of small nodular prominences, with thickening of the valve. The vegetations are much firmer than in the acute disease.
2. Formation of yellowish, opaque, fatty patches.
3. Great increase of fibrous tissue, which subsequently contracts, producing much deformity. The cusps become rigid, curled, and may cause great obstruction to the onward flow of blood, and at the same time fail accurately to close together when required.
4. Great narrowing of the valvular *orifice*.
5. Shortening of the chordæ tendineæ and papillary muscles. Frequently fusion of the chordæ tendineæ (adhesions).
6. Calcification of the fibrosed portion.

EFFECTS OF CHRONIC VALVULAR DISEASE.

We have seen that as a result of inflammatory affections very serious structural changes occur in the valves of the heart. We must, however, consider in detail the effects of such morbid changes. The points which must ever be borne in mind are that normally—

1. By means of the pumping force of the heart the tissues generally are supplied with oxygenated blood necessary for their perfect nutrition, and to enable the various organs to carry out their functions.

2. The perfection with which organs carry out their functions depends upon the amount of oxygenated blood *passing through them in a given time*.

3. In order, therefore, for the heart to do that which is required of it, it must be perfect in its structure and properly nourished.

We can then have untoward and down-grade changes brought about by—

- (1) Interfering with the heart's nutrition.
- (2) Valvular defects.
- (3) Changes in the innervation.

We shall study these changes in detail, but meanwhile let us bear in mind three other important points.—

1. The normal cardiac mechanism is adapted to meet a certain amount of sudden strain.

2. This reserve power is developed, and *actually increased under increased strain, provided the heart is adequately supplied with blood, and the strain gradually applied.*

3. Notwithstanding this grand reserve, a time comes when reserve force must fail, and symptoms of heart failure develop—in other words, hypertrophy keeps up the balance for a time; but ultimately dilatation becomes excessive, the down-grade process commences, to end finally in complete failure. Let us take for an example “aortic stenosis.” The first effect will be an extra strain upon the valve and chamber behind (left ventricle). Under the extra strain the chamber at first dilates *slightly*, as the ventricle has no previous preparation for the sudden establishment of a lesion in front of it. But the reserve energy of the myocardium is soon called into play, the ventricle contracts more forcibly to overcome the obstruction, and its walls begin to hypertrophy. As the obstacle is always present, the hypertrophy increases daily until in course of time it may come to be very great. The obstruction to the onflow of blood is thus overcome by more forcible and more prolonged contractions, the arterial system remains well supplied, and for a time no bad symptom may develop. This is known as the “Stage of Compensation” of the valvular lesion. Finally, however, the reserve energy of the ventricle becomes exhausted owing to prolonged overwork, aided perhaps by external causes such as faulty regulation of habits, diet, or exercise, or mental worry and anxiety. Then the exhausted muscle yields to the strain, and dilatation takes the place of hypertrophy. The walls of the ventricle are carried apart, and in consequence the

curtains of the mitral valve, though healthy, are no longer able to close the orifice ("*relative insufficiency*"). Regurgitation of blood into the auricle (mitral regurgitation) follows, and upon that engorgement of the pulmonary circulation.

The right ventricle is now called upon to force the blood through the engorged lungs to the left side of the heart, and it must obviously pass through changes similar to those that affected the left ventricle. But, as it is a less muscular structure, these changes are completed sooner. Relative tricuspid insufficiency is established, and with it the last stage, known as "failure of compensation."

The aortic symptoms *first* developed may thus *become masked by mitral symptoms*, although death may ensue before these appear.

Evidently, in cases of disease of the mitral valve, causing either back-flow of blood into the left auricle or obstruction to the passage of blood from the auricle into the left ventricle, the right ventricle is the compensatory force, and will therefore undergo hypertrophy early, while the left ventricle is either not hypertrophied, or only moderately so.

The duration of compensatory hypertrophy, in each individual instance, depends *mainly* upon the extent to which the myocardium has been involved in the original endocarditis.

It must be borne in mind that chronic valvular lesions are often produced not as the result of endocarditic changes whether acute or chronic, but as the result of degenerative changes such as atheroma. These latter arise as a rule at a later period of life than rheumatic endocarditis, although the onset of atheroma may be hastened by alcoholism, syphilis, or prolonged overstrain. Atheromatous change affects the aorta and spreads thence to the curtains of the valve. It may afterwards extend, but not very often, to the mitral valve. Rheumatic endocarditis attacks the mitral valve most frequently, although it may implicate the aortic also, or attack it singly. Aortic lesions then often originate in middle life, mitral lesions as a rule in early adult life.

FAILURE OF COMPENSATION.

Upon the establishment of relative tricuspid insufficiency there follows at once regurgitation of blood into the right auricle (tricuspid regurgitation), which becomes engorged, and offers an obstacle to the return of blood from the systemic veins. As the mitral lesion led to engorgement of the pulmonary, so does the tricuspid lead to engorgement of the systemic circulation. There is thus established a state of *passive congestion* of the great internal organs and of the subcutaneous circulation, and also, in extreme cases, of the serous cavities.

The *physical signs* of failure of compensation are of two classes :—

I. Direct signs of tricuspid regurgitation.

1. Systolic murmur in tricuspid area.
2. Weakness of second pulmonic sound (part of the blood which should flow into the pulmonary artery regurgitating into the right auricle).
3. Marked irregularity of the pulse.
4. Venous pulse, best seen in the neck (there being no valves between the jugulars and the incompetent tricuspid valve), and also in the liver (hepatic pulse).

II. Signs of passive congestion.

1. Œdema, beginning where the circulation is normally at the greatest disadvantage, *i.e.*, in the feet and ankles, and extending gradually upwards till the trunk and arms may be affected.
2. In grave cases ascites, hydrothorax, hydropericardium.
3. Congestion of internal organs :—
 - (a) Liver. Enlargement, tenderness, slight icteric tinge.
 - (b) Kidneys. Scanty high-coloured urine, abundant urates, albumin in varying amount, a few hyaline casts and isolated red blood corpuscles. Urea not diminished.

(c) Stomach. Dyspepsia, catarrh, often hæmatemesis.

After this extended explanation it will be easy to follow the course of events, which is really *physiological* once the diseased process has been established, in each individual valvular lesion. These will, therefore, be only briefly dealt with.

HYPERTROPHY OF THE HEART.

We have already seen that hypertrophy of the heart is a natural sequence of increased vascular strain, however brought about, *provided the heart muscle itself receives a sufficient blood supply to keep up its nutrition.*

Causes.—

1. Secondary to valvular lesions.
2. Adherent pericardium.
3. Secondary to diseases of the lungs.
4. As a result of increased peripheral arterial resistance—
 - (1) Obstruction of arteries by pressure of morbid growths.
 - (2) Atheromatous degenerations, or arteriosclerosis, as seen in Bright's disease, gout, etc.
5. Aneurism of the aorta, etc.
6. Over-exertion of a healthy heart, as seen in soldiers, hammermen, professional runners, etc.
7. Long-continued functional excitement.

It will be readily understood that hypertrophy is a compensatory change—*i.e.*, increased growth to meet increased work. But, whilst hypertrophy *per se* is a beneficial condition, it must also be regarded as a distinct weakness; for the patient has already called upon his normal reserve force; and a time comes when the nutrition becomes inadequate, dilatation replaces hypertrophy, and then compensation fails. The man with a hypertrophied heart may be compared to a country menaced and irritated by an enemy; and in order to prevent actual war the reserve forces are called out to supplement the standing army, which causes an additional drain upon the country's exchequer.

Signs of Hypertrophy—

1. Bulging of the præcordia, when the disease has begun in early life. If after complete ossification, bulging uncommon.

2. Alterations in the apex beat.

(1) Its *visible* area is largely increased.

(2) It is slow and heaving.

(3) The apex beat may be *felt* in the sixth, seventh, or even eighth interspace *outside* the nipple.

The above, of course, are the signs of hypertrophy of the *left ventricle*.

Hypertrophy of the *right* ventricle is usually due to lung disease, mitral obstruction, mitral regurgitation, or a combination of the two. For years the effects of mitral regurgitation *per se* may be counterbalanced by perfect hypertrophy.

Symptoms of right ventricular hypertrophy.

1. Bulging of the lower part of the sternum.

2. Diffuse "apex" beat. The beat is mainly due to the pulsation of the right ventricle under the chest wall, the apex being displaced to the left and under the lung.

3. Moderate increase in the cardiac dulness towards the *right*.

4. Accentuation of the second sound in the pulmonary area, due to increased tension in the pulmonary artery.

The degree of hypertrophy of which any section of the heart is capable is largely determined by the age of the patient. If one submits to the same course of gymnastic exercises a lad of eighteen and a man of fifty, the increase in volume of the muscles is much more apparent in the former. So with the heart; suppose (*ceteris paribus*) two cases of aortic regurgitation, the one commencing at eighteen, the other at fifty. In the former the hypertrophy of the left ventricle will be very great and long maintained; in the latter the ventricle is no longer capable of great hypertrophy, and relative mitral insufficiency is likely to be sooner established.

DILATATION OF THE HEART.

Under Failure of Compensation we have already considered the main points in dilatation of the heart. It only remains for us now to consider the more minute changes that occur in this condition. Whilst undoubtedly the main factor in the production of dilatation is *obstruction*, either through valvular disease or increased peripheral resistance, still, we must remember that dilatation is hastened and even brought about by degenerative changes in the ventricular *walls*, such as—

1. Fatty degeneration.
2. Fibrosis.
3. Anæmia.

High temperature has already been referred to as a frequent cause of dilatation, on account of the changes it induces in the cardiac musculature.

Anatomical Changes.—The heart is more globular in shape. The walls of the affected ventricle are much thinned, especially at its apex. The auriculo-ventricular valves are usually markedly incompetent.

Physical Signs.—

In dilatation of the right ventricle—

1. Great increase of præcordial dulness to the right, and to a less degree to the left.
2. Diffuse pulsation over præcordia.
3. Feebleness of apex impulse and heart sounds.
4. Systolic murmur in tricuspid area.
5. Irregularity of pulse.
6. Venous pulse.

Symptoms have been summed up under Failure of Compensation.

VALVULAR LESIONS.

We shall have to reiterate much that has already been said under endocarditis. We have already seen that each valve may be affected in one of three ways, viz.—

1. They may be narrowed, impeding the flow of blood—obstruction or stenosis.
2. They may be incompetent—regurgitation or insufficiency.
3. They may be incompetent and obstructed.

These conditions are diagnosed by—

- (1) The position, rhythm, and direction of propagation of the murmurs accompanying such conditions.
- (2) The general symptoms produced by such lesions.

Murmurs are propagated in the direction of the blood-stream, and are best heard a little way from the valvular orifice where they are produced.

Always in describing a murmur, state—

1. Position.
2. Time.
3. Propagation.
4. What sound (if any) is modified thereby.
5. Accompaniments (if any).

Examples—

Aortic Stenosis.—A murmur heard in the aortic area, systolic in time; propagated up the sternum into the carotids, and usually *accompanied* by a loud first sound.

Aortic Regurgitation.—A murmur heard in the aortic area, *diastolic* in time; propagated *down* the sternum, and modifying or replacing the second sound.

In both conditions there are hypertrophy of the left ventricle, greater in regurgitation; anæmia of the systemic circulation and tendency to syncope, greater in regurgitation; and very often cardiac pain of an anginoid type, or true angina pectoris.

Mitral Stenosis.—A murmur heard in the mitral area, *pre-systolic* in time; *runs up to the* first sound—not propagated, but often accompanied by a marked presystolic thrill (purring tremor).

Mitral Regurgitation.—A murmur heard in the mitral area, systolic in time; propagated to the axilla, *and modifying or replacing* the first sound. If the murmur be loud, it may be heard at the back, close to the left side of the spine.

In both conditions the symptoms, before compensation has failed, are mainly pulmonary—increased liability to bronchial affections, dyspnoea, cyanosis, hæmorrhage, œdema of lung. Pain is not usually severe, though there may be a sense of emptiness or cardiac distress.

Tricuspid Regurgitation.—A murmur heard over the fourth right costal cartilage and lower part of the sternum; propagated to the right and slightly to the left; systolic in time.

Pulmonic Stenosis (the commonest of the congenital lesions).—A murmur heard in the second left interspace, close to the sternum; systolic in time; propagated upwards. The murmur is often very loud, the right heart is enlarged, and cyanosis is marked.

Other murmurs are too rare to require a description in such a book as this. Too much importance may easily be attached to the mere localisation of murmurs, if *little* notice is taken of the state of the heart nutrition. Post-diastolic and mid-diastolic murmurs are often diagnosed with lightning rapidity by the student, who, at the same time, fails to note whether compensation is established or beginning to fail.

Reduplication of sounds indicates a want of synchronism between both sides of the heart. Remember, the second sound is produced by the closure and stretching of the aortic and pulmonary valves; the first sound, by closure and stretching of the auriculo-ventricular valves, *plus the contraction of the ventricular muscle.*

Lastly, do not forget that the loudness of a murmur is but a poor index of the gravity of the lesion. A careful consideration of the general state of the heart and *general symptoms* is of far greater importance than mere attention to any murmurs that exist. The diagnosis of cardiac lesions is sometimes extremely easy, and at other times very difficult. Aortic disease may be masked by mitral disease; mitral disease by

extensive pulmonary disease, and so on. Remember that the tendency of all organic cardiac lesions is *finally to produce arterial emptiness and venous congestion*. The following table will show the more important differences.

AORTIC DISEASE.	MITRAL DISEASE.
Symptoms are mainly due to anæmia, viz.—	Symptoms are mainly due to venous congestion, viz.—
1. Pallor.	1. Cyanosis.
2. Throbbing of the carotids.	2. May get pulsation in the veins of the neck.
3. Attacks of an “angina pectoris” type of pain.	3. Sudden attacks of severe dyspnoea—actual acute pain is rare.
4. Breathlessness on slight exertion.	4. Breathlessness, but often present without exertion.
5. Menses usually absent.	5. Often there is menorrhagia.
5. Nervous symptoms are prominent, owing to cerebral anæmia. Syncopal attacks are also common.	6. Pulmonary symptoms are most prominent, <i>i.e.</i> —
	(1) Cough in the morning.
	(2) Chronic Bronchitis.
	(3) Hæmoptysis.
	(4) Dilated right side of heart, and later—
	(5) Symptoms due to tricuspid leakage.
7. The left ventricle is usually much hypertrophied.	7. The left <i>auricle</i> is somewhat hypertrophied, but the main hypertrophy is that of the right ventricle.

Tricuspid Disease.—The symptoms of failure of the right side of the heart may be summed up as—those symptoms which always arise from imperfect aeration of blood, and venous congestion of the organs generally. Those in connection with the congested organs are—

1. Lungs = dyspnoea, bronchitis, pulmonary œdema.
These are the result of obstruction to the pulmonary circulation caused by the disease on the left side.
2. Liver (nutmeg) = dyspepsia, hæmatemesis, ascites.
3. Kidney = albuminuria, general dropsy, uræmia.
4. Skin = œdema.

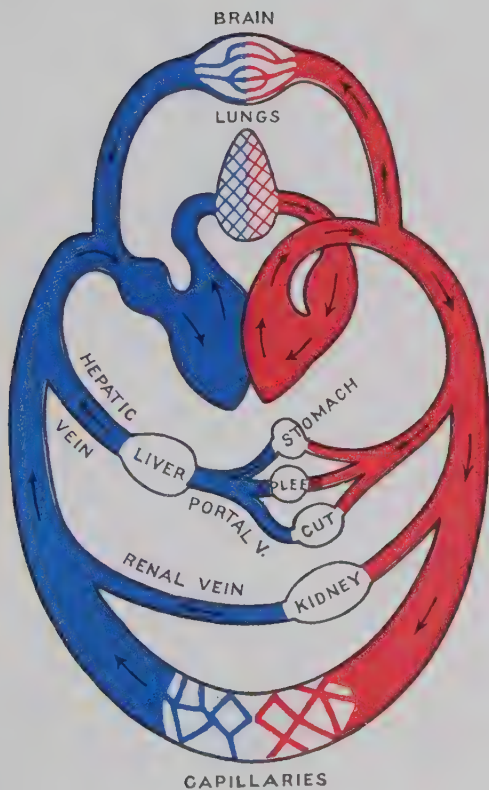


DIAGRAM for studying the results of backward pressure. Note the areas in blue will become the seat of changes consequent on venous congestion.

The symptoms are, in fact, merely an exaggeration of those arising from mitral disease, to which it is usually secondary. When primary, it is generally a congenital lesion. It may of course arise out of emphysema. The patient finally becomes water-logged. Locally there is epigastric pulsation. The jugular vein, if obstructed by the finger, *fills up from below during systole*.

Besides the single lesions just described, there may be various combinations of valvular lesions. Probably the most frequent is a combination of stenosis and regurgitation affecting the same valve. It is quite rare to meet with aortic stenosis without some degree of aortic regurgitation, and even in mitral stenosis there is usually a regurgitant element, though it may be only slight. Single lesions, proved so *post mortem*, are generally regurgitant. There may be combined lesions of different valves, as aortic regurgitation and mitral stenosis and so on.

THE PULSE IN CARDIAC LESIONS.

Aortic Stenosis.—Here the blood is obstructed in its flow into the aorta. The pulse is thus small, and the sphygmographic line of ascent is slow, and often interrupted (anacrotic). The tension will depend on the amount of obstruction and degree of hypertrophy. The lesion is seldom solitary, but is usually accompanied by some degree of regurgitation, which modifies the characters of the pulse.

Aortic Regurgitation.—The ventricle in this condition is filled during diastole by *two* streams—one the regurgitant aortic stream, and the other in the usual way from the auricle. The chamber is thus filled more quickly, and the pulse made to beat faster. The sudden rise and the sudden fall of the pulse give it a peculiar kicking or water-hammer character. This is due to the collapsed state of the arteries between pulsations, owing to regurgitation into the ventricle, and their sudden filling with each forcible systole. The throbbing of the carotids often prevents sleep. *Capillary pulsation is often well marked*.

In *Mitral Disease* the pulse is often small and of low tension. After failure of compensation has set in, irregularity in force and rhythm is its chief feature, but in mitral stenosis it may be irregular even before compensation is permanently disturbed.

Prognosis—

Aortic Stenosis.—Prognosis is comparatively favourable, as hypertrophy is usually well marked and maintained. Often there are no symptoms suggestive of such a grave condition for some considerable time. When stenosis is solitary it is often attended by, and due to atheroma, and the fragile state of the vessels may lead to cerebral hæmorrhage.

Aortic regurgitation on the other hand is a much more dangerous lesion; sudden syncope through anæmia of the brain is often fatal. Angina pectoris is common in this lesion and may cause speedy death.

Either lesion may go on to dilatation of the left ventricle and implication of the right side of the heart, thus ending like mitral disease, or the exhausted ventricle may cease to beat before the right side is materially affected (death by asystole).

When both lesions exist in the same case, the stenotic lesion may be regarded as in a sense protective. Evidently, when the aortic orifice is narrowed, less blood must flow back into the ventricle during diastole, and hence the arteries are not so completely emptied.

Mitral disease is characterised by frequent breakdowns, but patients often quickly recover their usual condition and may be long-lived, especially in cases of mitral regurgitation. The prognosis in mitral obstruction is graver, as compensation breaks down earlier. In women it is especially apt to be fatal during pregnancy and the puerperal period.

Tricuspid disease is always grave, and it is impossible to give any general prognosis. Each case must be carefully considered on its own merits.

In all lesions of the left side of the heart the possibility of embolism must be kept in mind, the most serious being, of course, embolism of the brain, with its resultant hemiplegia.

Treatment.—It cannot be too clearly understood that there is no *routine* treatment for valvular diseases of the heart. Although digitalis is to be looked upon as the typical cardiac tonic, and must be used at some stage in the majority of cardiac lesions, yet its indiscriminate use may be productive of much harm by causing excessive stimulation of the muscle. Similarly, in the same cases, much benefit may be obtained at one time from rest, at another from carefully graduated exercise. Certain general indications, however, point towards the selection of a particular line of treatment.

1. Hypertrophy is nature's cure ; and, in order
2. To get hypertrophy there must be a wholesome supply of blood to the heart tissue.
3. Remember the golden rule "avoid putting excessive strain on a diseased tissue."
4. Don't look upon the heart as an isolated structure, but as a *component and essential part* of a complex machine ; and consequently sharing in the anabolic and catabolic changes of the body generally. In other words, if the system generally be lowered, then the heart must also suffer ; but this does not prevent us from using those drugs which have a distinct and particular action upon the heart. Our treatment must be then—

GENERAL AND MEDICINAL.

(1) *Diet.*—Avoid excesses, especially of nitrogenous food, which tends to increase *peripheral resistance*. It is often advisable to limit the amount of food taken at any one meal, and to give small meals often, rather than large meals seldom. Heavy meals before bedtime are to be avoided.

NOTE—A full or distended stomach is a constant source of cardiac embarrassment.

Where compensation has failed, the diet should at first be fluid, milk being the staple food.

(2) *Exercise.*—Provided the *assimilative powers are good and plenty of food can be taken to provide for the increased*

expenditure of energy, then graduated exercise must be good, as it initiates or helps the natural way of producing hypertrophy of the chambers behind the lesion ; on the other hand

(3) *Absolute rest is the treatment* when the assimilative powers are weak, or compensation once established is failing.

(4) *Drugs* include (1) general tonics, and (2) those having a special action on the heart. In many instances of established compensation treatment by drugs is almost unnecessary, careful regulation of the patient's diet and habits sufficing to maintain him in relative health. In most instances it is well to inform him of his condition, at the same time reassuring him and pointing out the need for caution. In this stage, if drugs are needed, they are general tonics, such as iron, arsenic, or strychnine.

Where compensation has failed, cardiac tonics are required, and of these digitalis stands first. It acts beneficially by slowing the action of the heart and prolonging its diastole, thus increasing the natural rest. It also increases the force of the cardiac beat. In addition it has a diuretic action and thus tends to relieve dropsy. Its disadvantages are that it increases resistance in the peripheral arteries and so produces greater strain, and that in aortic regurgitation the prolonged diastole permits of a longer back-flow. Do not give digitalis, though there be cardiac disease, if compensation is well established ; and, secondly, do not continue giving digitalis if the urine decreases in quantity after its administration, or if the pulse tends to become more irregular. To avoid its cumulative effect, it should not be given for more than three weeks to a month at a time. It may be resumed after an interval of a week or ten days, during which other cardiac tonics may be used if necessary.

Strychnine is always a powerful adjuvant to digitalis, and is the best cardiac tonic in cases where compensation has not broken down.

Of other cardiac tonics strophanthus holds the second place, and may be used where digitalis disagrees, or in combination with it. Sparteine, convallarin or the tincture of convallaria

majalis, casca, etc., are also frequently employed. Sudden breakdowns of compensation call for stimulants such as ether, ammonia, brandy mixture, *complete rest*, cupping over the lungs, etc.

Cardiac pain is best relieved by morphine and iodide of potassium. If there be arterial constriction, nitrite of amyl is the best remedy.

Where there is much œdema and congestion of internal organs diuretics may be used in conjunction with digitalis. Absolute rest in bed and milk diet will also help to relieve the condition. Purgatives are not indicated. The subcutaneous tissues may be drained by acupuncture or by Southey's tubes.

A CAUTION.—If you are called to see a patient who has been picked up insensible, and smells strongly of alcohol, do not hastily conclude he is drunk, but *make sure of the state of the heart*. Many patients finding themselves getting faint, take a dram of brandy, which, however, may fail to prevent syncope. Rough usage or a cold cell would probably kill a patient under such circumstances.

FUNCTIONAL DISORDERS OF THE HEART.

The functional disorders include—

1. Syncope—to be carefully distinguished from the *petit mal* of epilepsy.
2. Palpitation—*i.e.*, forcible and often irregular action of the heart, perceptible to the individual.
3. Arrhythmia, or intermittent condition of the cardiac beats.
4. Tachycardia or rapid heart.
5. Bradycardia or slow heart.

[For the details of these conditions the reader is referred to a larger text-book.]

Rapidity of the heart is usually associated with a neurotic temperament, menstrual epochs, excessive use of stimulants, mental excitement, use of certain drugs, such as belladonna, etc.

Slowness of the heart is most commonly associated with

hunger, the puerperal state and melancholia, use of digitalis, excessive use of tobacco, poisoning *by bile, lead, etc.* Often, however, the same conditions will produce rapidity in one individual, and slowness in another.

Such conditions may obviously also occur in organic diseases of the heart, but if they constitute the chief complaint, they are *prima facie* more likely to be functional. Patients very often imagine themselves to have cardiac disease on account of palpitation, etc., induced by gastric or other non-cardiac derangement.

PAROXYSMAL BRADYCARDIA.

(STOKES-ADAMS DISEASE.)

A condition characterised by paroxysmal infrequency of the pulse associated with syncopal or epileptiform attacks.

Ætiology.—The disease is commonest in males over fifty years old. It is generally associated with cardiac disease due to lesions of the coronary arteries. Fibroid degenerations of the myocardium are thus produced. In several instances such degeneration has been found to affect the recently described auriculo-ventricular bundle of His, through which the auricular impulse is supposed to be transmitted to the ventricles.

Symptoms.—There may be evidence of valvular lesions, especially of the aortic valve; or there may be merely evidence of dilatation and cardiac failure. The arteries are often tortuous and rigid. The patient is usually pallid, but the nose and finger-tips may be cyanosed. The pulse is persistently infrequent, but in the paroxysms falls to a very low rate (20 per minute or less). The pulsation of the auricles is often more frequent than the ventricular pulsation (compare the jugular pulse with the apex beat).

Dyspnoea is common, and may amount to cardiac asthma. *Cheyne Stokes breathing* may occur in the paroxysms. In the paroxysms, too, *nervous disturbances*, varying from mere giddiness or transient loss of memory to unconsciousness (syncope) or even epileptiform attacks, are frequently present.

Pain is not a constant feature, but there may be anginoid attacks.

Such cases end fatally, but life may be prolonged for several years. The end is usually sudden.

Treatment.—Exercise and diet must be regulated according to the individual case. Iodides are the most reliable medication, and may be combined with nitrites if there is much arterial spasm. Digitalis and strophanthus must be used with caution. They have no effect upon degenerated cardiac muscle, and digitalis especially may dangerously increase peripheral tension. During the attacks diffusible stimulants are necessary. If there be marked dyspnoea or Cheyne-Stokes breathing, oxygen, or hypodermic injections of strychnine in large doses, may tide over the crisis.

ANGINA PECTORIS.

(“Breast Pang.”)

A condition characterised by a sudden severe pain in the chest, with a sense of impending death.

Ætiology.—It occurs most frequently amongst men above the middle age. Predisposing causes are “*all conditions which interfere with the nutrition of the walls of the heart*”; such as extensive fatty disease, valvular diseases, atheroma, however brought about, obstruction of the coronary arteries, etc., also affections of the nerves of the cardiac plexus, or the intrinsic nerves and ganglia of the heart; the exciting causes are:—

Sudden strain, an over-distended stomach, powerful emotional disturbances, etc. John Hunter said of himself, “My life is in the hands of any rascal who insults me.” He died of angina pectoris, induced by a fit of anger.

Symptoms.—The patient is suddenly seized with an acute and sudden pain across the chest. If he is walking, he stops rigid and motionless; if he is sitting or in bed, he leans forward, fixing the shoulder girdle by grasping any convenient support. The terrible feeling of anxiety is reflected in his expression.

The pain is most marked at the lower end of the sternum and radiates down the *left* arm most frequently, though the *right* arm is often implicated. The patient feels as if an iron band was fixed around the chest. The feeling of suffocation is intense, but the usual cyanosis of dyspnoea is absent, the face often being extremely pale. Respirations are very shallow and difficult, though there is no obstruction to the entrance of air. A sphygmographic tracing of the pulse taken during an attack shows increased arterial tension. An attack may last from a few seconds to many minutes; may kill the first time, or recur at various intervals.

Pathology.—*Post-mortem*—the heart is usually distended with blood, and the walls and valves often show extensive morbid changes. The coronary arteries are extensively diseased. There are, however, cases in which no organic disease is discoverable (the so-called *angina pectoris vasomotoria*). In these a widespread constriction of the peripheral arteries may lead to secondary embarrassment of the left ventricle.

Causation of the Attack.—Many people have extensive heart disease without getting angina. Again, a patient may have only one attack, or have an interval of years between them, so, *per se*, heart disease is *not sufficient to produce angina*. We must confess that at present the explanations of the condition given are not altogether satisfactory. The following statements possibly show the extent of our knowledge at present. Atheromatous coronary arteries cause—

1. More or less anaemia of the heart's substance.
2. Irritability of the intra-cardiac ganglia.
3. Diminished contractile power of the ventricles.

Let us suppose a heart with all these disadvantages is subjected to a severe strain. What will result?

1. Over-distension of the ventricle.
2. A reflex message will be transmitted from the irritable ganglia to the medulla, resulting in sudden *general vasomotor constriction*. Therefore, the heart *already* embarrassed has to pump against *increased*

peripheral resistance, hence the feeling of suffocation, etc.

The pain, then, is produced by a combination of distension from within and spasm of the muscular substance of the peripheral arteries.

If this be true, then the use of nitrites is at once apparent, for, as they cause dilatation of the arterioles, the peripheral resistance *is at once diminished*, and the over-distended ventricle allowed or enabled to empty itself. Indeed, the only flaw in the foregoing explanation is, "that in some instances of angina no increase of arterial tension has been observed." This is but a poor argument though, for we can easily understand that the propelling force of the heart may be insufficient to cause any *appreciable tension*, notwithstanding the increase of peripheral resistance.

Treatment.—During the attack administer nitrites, preferably by inhalation; chloroform may be used as a substitute, or, if the nitrites fail, morphine. During the interval, careful attention to the bowels, diet, and avoidance of severe mental or muscular exercise, with the administration of iodides, considerably lessens the chance of *another attack*. Nitroglycerine, given in doses of m j of the 1 per cent solution thrice daily, and gradually increased if necessary, is often of great benefit. Lastly, it must be remembered that many patients complain of something like angina, which, however, is not the true disease.

TRUE ANGINA.

Most common past middle life.
Most common in men.
Attacks—rarely nocturnal or periodical.
Not associated with other symptoms.
Agonising pain and sense of constriction.
Pain of short duration.
Lesions of arterial sclerosis.
Prognosis grave; often fatal.

PSEUDO-ANGINA.

At every age from six years.
Most common in females.
Often periodical and nocturnal.
Associated with *nervous* symptoms.
Pain less severe—distension more than constriction.
Pain lasts one or two hours.
Neuralgic affection.
Never fatal.

(Huchard's table).

ATHEROMA.

Atheroma is the most common abnormality of the arteries. Some degree of it is almost invariably present in advanced life. It may occur earlier as a result of hereditary predisposition. Other predisposing causes leading to its early development are alcoholism, syphilis, gout, and plumbism.

Anatomy.—In this affection the tunica intima is thickened and its deeper part undergoes, in irregular patches, fatty or atheromatous degeneration. The degenerated portions become infiltrated with lime salts, forming nodules or large plates according to the size of the artery. The degenerated patches may also become cystic, and the cysts may rupture through the intima, leaving an atheromatous ulcer. Or the calcareous plates may crack, and lacerate the intima. Ultimately the muscular coat atrophies or becomes fibrosed.

Signs of Atheroma.—Rigidity, visibility, and tortuosity of the peripheral arteries,—radial, brachial, carotid, temporal, etc. The wall of the vessel can be felt after obliteration of the pulse. Irregular thickenings are to be felt in the wall, and the edges of the calcareous plates are often clearly palpable. The pulse is visible.

Atheroma may be far advanced in the great vessels (especially the aortic arch) when there is little sign of it elsewhere.

Results.—Cerebral arteries—aneurism, rupture, or thrombosis; coronary arteries—narrowing, thrombosis, angina pectoris; arteries of limbs—thrombosis, senile gangrene; arch of aorta—aneurismal dilatation or sacculated aneurism, also extension to cusps of valve, with aortic stenosis or regurgitation or both.

ANEURISM.

Before discussing the essential features of thoracic aneurism we must recall a few facts relative to *aneurisms in general*. An aneurism may be defined as “a localised and persistent dilatation

of a blood-vessel." Surgeons employ the term in a wider sense. Classified according to shape we have the following varieties—

1. Fusiform or spindle-shaped, involving the *whole* circumference.
2. Sacculated—where one side of the vessel only is dilated.
3. Diffuse—where a large portion of the vessel is irregularly dilated.

Cause.—The two main factors are—

1. Damage to the vessel walls.
2. Increased vascular strain.

1. Damage to the vessel wall is due to arterio-sclerosis, atheroma, syphilitic arteritis, or causes acting from without, as pulmonary tubercle.

2. Increased vascular strain is the result of laborious occupations (hammermen, riveters, soldiers subjected to forced marches with heavy kit, etc.).

It is possible that even a single very violent overstrain may rupture the coats of a previously healthy artery. At least a history of "something giving way" within the chest, as a result of such strain, is not uncommon.

Pathology.—

The inner coat may be much thickened by atheromatous changes, but much more frequently it disappears altogether, a delicate layer of hyaline tissues taking its place.

The muscular coat.—The muscular fibres become stretched and dissociated, and ultimately disappear.

The outer coat becomes much fibrosed, and adherent to the surrounding tissues by an inflammatory process; thus the "sac" of an aneurism is most frequently composed of dense fibrous tissue.

Course.—An aneurism once started tends to increase, and, if not arrested, finally ruptures. But an aneurism may cure itself by becoming so large that the sac, by pressing on the artery, checks or even obliterates the blood supply. Again,

when the orifice is small in a sacculated aneurism the circulation in the sac may be so impeded that thrombi form, ending in a large pale laminated clot, which may ultimately organise and transform the aneurism into a fibrous nodule. This occurs, however, only in the smaller aneurisms; but this natural cure gives the physician the cue as to what he should attempt.

Symptoms.—The cardinal symptoms of an aneurism are—
(1) Tumour. (2) Pulsation. (3) Systolic murmurs. (4) Pain.
(5) Other pressure effects.

The extent of these symptoms depends upon the site of the tumour and the nature of the adjacent structures. A word of caution is needed to guard the beginner against diagnosing *an aneurism of the aorta too quickly*; frequently there are no symptoms indicative of such a serious condition, and on the other hand there are often grave and suggestive symptoms present, but *no* aneurism. We shall now consider the special points of

THORACIC AORTIC ANEURISM.

The aorta is the most frequent seat of aneurism in the body, and all varieties have been found here. There are many reasons given why aneurisms should be so common in the arch of the aorta—

1. It is much curved.
2. The first part of the arch has very little support.
3. The blood-stream ejected during systole of the heart tends to bulge the aorta locally.
4. This part is much more affected by the variation of the cardiac pressure than the distal arteries.
5. It gives off large branches in a very small area.
6. The vessels tend to dilate during inspiration.
7. Aortitis and atheroma are very common in this situation.

Occurs most frequently amongst men who are either prematurely old through intemperance, syphilis, etc.; or in those

engaged in occupations which tend to increase the normal aortic strain, such as hammermen, brewers, young soldiers, etc. Septic emboli lodging in the inner coat account for some cases. Traumatism may also cause aortic aneurism.

Symptoms.—They depend on the portion of the arch affected, and size and shape of the aneurism.

Briefly they are—

1. *Symptoms in connection with the Circulation.*—Palpitation, angina pains, the arteries are filled less perfectly, and there may be a difference in the two radial pulses.

2. *Symptoms due to Pressure*—

(1) *Œsophagus.*—There is difficulty in swallowing, especially solids.

(2) *Respiratory System.*—Less air enters the lung pressed upon, and the breath-sounds are consequently weak. There is much dyspnœa, or attacks of the so-called aneurismal asthma, a peculiar alteration of the voice and cough (leopard growl and gander cough). Hæmoptysis may occur later due to “weeping” of the aneurism.

(3) *Implication of Nerves.*—The symptoms will depend on the amount of pressure exerted on the nerves. Thus, if *slight*, we get symptoms due to *irritation*; if *severe*, symptoms due to *paralysis*. The nerves most likely to be involved are—

	IRRITATION.	PARALYSIS.
<i>Left Recurrent Laryngeal</i>	Alteration of voice; and stridor, due to spasm.	Aphonia. Paralysis of left vocal cord.
<i>Phrenic</i>	Painful and persistent hic-cough.	Intercostal breathing. Death.
<i>Sympathetic</i>	Dilatation of the pupils. Pallor from constriction of the vessels on the one side. Rapid action of the heart.	Contraction of the pupil and flushing of that side of the face.
<i>Vagus</i>	Depressed action of the heart. Vomiting and nausea.	Irregular action of the heart. Pneumonia. Death.

Veins.—Edema of superior extremities, one side of the head, etc.

Thoracic Duct—produces rapid emaciation, and fatty stools.

Bones—are eroded and absorbed; the process being accompanied usually with intense boring pain. When the spine is involved the pain is intense, due to irritation of the intercostal nerves and meninges—there may be much deformity, and even paraplegia through implication of the spinal marrow—a murmur may be heard over the spine.

Such then are the clinical symptoms; on

Physical Examination—

Inspection may reveal a swelling; or there may be a diffuse heaving impulse above the third right costal cartilage, and displacement of the apex beat. If the descending part of the arch is affected there may be bulging to the left of the spine.

Palpation often detects the expansile character of the tumour, with its *systolic* thrill and diastolic shock.

Percussion.—Note is dull or flat and gives a feeling of resistance.

Auscultation may reveal, over the dull area, a ringing accentuated second sound and a systolic bruit.

Dr. Osler calls attention to the absence of pulsation in the abdominal aorta or the femorals in cases of large thoracic aneurisms.

If the chin be brought forward to relax the neck, and the cricoid drawn upwards, there is felt a peculiar downward tugging at each systole if the aneurism be attached to the bronchus or trachea (*tracheal tugging*). The same feeling is not produced by any other tumour.

According to the part of the arch involved, the physical signs and pressure symptoms may vary. The following table shows the chief differences:—

[TABLE

PART INVOLVED.	ASCENDING.	TRANSVERSE.	DESCENDING.
<i>Physical Signs</i>	<p>Pulsation, often expansile, in 2nd and 3rd interspaces. On palpation systolic thrill and diastolic shock to right of sternum.</p> <p>Dulness to right of sternum, above cardiac area.</p> <p>Rough systolic murmur, loud clanging second sound. May have diastolic murmur from implication of aortic valve.</p>	<p>Pulsation in episternal notch.</p> <p>Systolic thrill in episternal notch.</p> <p>Dulness over manubrium sterni.</p> <p>Murmur more distinct over manubrium. Diastolic murmur rare.</p>	<p>Pulsation, if any, to left of spine.</p> <p>Absent.</p> <p>No dulness anteriorly, sometimes dull to left of spine.</p> <p>Murmur may be absent; when present systolic, to left of spine.</p>
<i>Parts liable to Pressure and Results of Pressure</i>	<p>Vena cava superior; dilated superficial veins, œdema of head and neck.</p> <p>Innominate artery; weakness of right radial pulse.</p> <p>Heart; downward displacement of apex.</p> <p>Ribs to right of sternum; pain.</p> <p>Right bronchus; defective respiration on right side.</p> <p>Right recurrent laryngeal (rarely); paralysis of right vocal cord.</p>	<p>Left innominate vein; œdema of left side of head and neck.</p> <p>Any branch of the arch; weakness of right or left radial pulse.</p> <p>Manubrium sterni; pain.</p> <p>Trachea or left bronchus; paroxysmal dyspnoea, altered cough, defective respiration on left side.</p> <p>Left recurrent laryngeal; paralysis of left vocal cord.</p> <p>Sympathetic; dilatation or contraction of pupil, usually left.</p> <p>Œsophagus; dysphagia.</p>	<p>Spinal column, and ultimately cord; dorsal pain, afterwards paraplegia.</p> <p>Left bronchus; defective respiration on left side.</p> <p>Left recurrent laryngeal; paralysis of left vocal cord.</p> <p>Left sympathetic (often); dilatation or contraction of left pupil.</p> <p>Œsophagus; dysphagia.</p> <p>Thoracic Duct; rapid emaciation sometimes chylous ascites.</p>
<i>Rupture may occur</i>	<p>Externally.</p> <p>Into pericardium.</p> <p>Into right pleura.</p> <p>Into right bronchus.</p> <p>Into superior cava.</p>	<p>Into trachea.</p> <p>Into one or other pleura.</p> <p>Into left innominate.</p>	<p>Into left bronchus.</p> <p>Into left pleura.</p> <p>Into œsophagus.</p>

It will be seen that in aneurism of the descending part the physical signs may be very few, or even entirely absent, and the diagnosis must then be made from pressure symptoms alone. In some cases of aneurism symptoms may be latent till death occurs from internal rupture.

Treatment.—

General.—Everything must be done to quiet the circulation, by the observance of absolute rest and abstinence from all alcoholic drinks, etc. The diet should be nourishing, but limited, and *the quantity of liquid taken reduced to a minimum.*

Medicinal.—Anodynes and sedatives are called for, but iodide of potassium in large doses daily seems the most efficacious of drugs. It is difficult to say how the iodide acts; it certainly does not lower the blood-pressure to the same extent as many other drugs which are *less* efficacious in benefiting the aneurismal condition. Possibly the beneficial effects are due to the iodides causing absorption of inflammatory material, which would otherwise induce sclerosis.

Local treatment is highly unsatisfactory. All methods aim at starting coagulation. Briefly, they are—

1. Introducing needles, and scratching the walls of the sac to form a rough surface, and so bring about coagulation.
2. Introduction of fine steel wire or horse hair.
3. Electrolysis.
4. Large injections (6 to 8 oz.) of a sterilised 2 per cent solution of gelatine in normal salt solution have also been used. The injection is made into the buttock, and repeated weekly till coagulation is induced. It is by no means free from danger, and several fatal cases have been recorded.

Considering the hopeless character of this affection it is more than cruel where pain is severe to withhold morphia, as some anti-opium fanatics would have us do.

ABDOMINAL ANEURISM.

Site.—Most common near the celiac axis. It may grow upwards and push the diaphragm before it, or forwards and project anteriorly, or backwards and erode the spine.

Diagnosis.—Palpation reveals a definite tumour with the characteristics of aneurism already described. The tumour may be better felt in the knee and elbow position.

Especially care must be taken in diagnosing this condition in pregnant or hysterical females; also in cases of tumours lying *over* the aorta. The expansile character of the pulsation is especially valuable.

Treatment.—As in thoracic aneurism. Compression under chloroform may be tried, though it is decidedly risky.

Prognosis.—Bad. Death may take place from—

1. Compression paraplegia.
2. Embolism of superior mesenteric artery.
3. Complete obliteration of the lumen by clots.
4. Rupture (oftenest into duodenum).

DISEASES OF THE DUCTLESS GLANDS AND BLOOD.

I. DISEASES OF THE THYROID GLAND.

MYXŒDEMA.

A DISEASE associated with atrophy of the thyroid gland, a peculiar condition of the skin, overgrowth of the subcutaneous tissue, and serious mental conditions.

Ætiology.—Most common in women between thirty and fifty-five years of age; hereditary factors appear to be occasionally present; the poorer classes are said to furnish the larger number of cases. The cause of the atrophy of the gland is unknown, although an extreme variety of predisposing causes has been tabulated. Diseases of menstruation and the puerperal state may account for the greater frequency in women, who are affected five times as often as men.

Pathology.—That the disease is dependent on atrophic changes in the thyroid gland is now certain, reasoning from the results of the removal of the gland on the one hand, and from the beneficial effects following the internal administration of the healthy thyroid.

Professor Horsley distinguishes three stages after removal of the thyroid gland in *monkeys*.

STAGES.	DURATION.	SYMPTOMS.	REMARKS.
1. <i>Neurotic</i> . . .	1 to 3 weeks.	Tremors, rigidity, dyspnœa.	Young monkeys often die in this stage.
2. <i>Mucinoid</i> . . .	3 to 7 weeks.	Commencing hebetude, and mucinoid degeneration of the connective tissue.	Older monkeys die at this stage if not treated.
3. <i>Atrophic</i> . . .	5 to 8 weeks.	Complete imbecility, atrophy of all the tissues.	Monkeys survive if kept in warm air bath.

The untoward symptoms were associated with enormous fall of blood-pressure and temperature.

It will, therefore, be seen that the presence of the thyroid is necessary to avert myxœdema. Possibly its normal functions are *to keep the blood pure and free from some pernicious products*, the nature of which has not yet been discovered, but which if retained cause myxœdema.

The morbid anatomical changes are most marked in the **subcutaneous tissues**—

1. Nuclear proliferation or formation of connective tissue all around the hair follicles and sweat-glands.
2. Increased deposit of subcutaneous fat.
3. Presence in the subcutaneous connective tissue of a gelatinous cement allied to mucin.
4. Formation of elastic œdematous swellings above the clavicles.

The **Thyroid Gland** is atrophied, often indurated, and shows scattered groups of cells, the remains of the normal vesicles.

Interstitial nephritis has been observed in some cases, but this is probably an accidental coincidence.

Symptoms.—Patient presents a heavy, stolid countenance. The face is swollen, and the cheeks present a diffuse red flush. The rest of the face is pale. The hair is scant, coarse, and brittle. She speaks in a heavy, slow, and thick manner (leathery voice). She exhibits great intolerance of cold—skin is enormously thickened, *dry*, and often scaly, but does not

pit on pressure. The hands are said to be spade-like. The temperature is always subnormal. The menses are irregular, and there is often a tendency to hæmorrhages. Teeth become carious. The gait is peculiarly clumsy. Bowels are constipated. Later, the mental condition becomes very grave; hallucinations, convulsions, and coma may occur. It must not be forgotten that often towards the end albuminuria may occur, and the skin may pit on pressure.

Treatment.—Guard patient against cold—give nourishing food.

Specific Treatment.—Injections of—

1. Emulsionised thyroid gland.
2. Extract of thyroid gland hypodermically.

These were the first forms in which the gland was used.

It was then shown that eating the cooked thyroid is equally efficacious, and now treatment is carried out by the administration of thyroid extract, either as the *Liquor Thyroidei* (B.P.), or dry, in the form of tabloids, etc. Small doses should be given at first, and gradually increased up to $\text{m} \times$ of the liquor twice daily. As the condition improves, the dose may be gradually lessened, but the patient must be told that she will have to continue the treatment all her life.

SPORADIC CRETINISM

Is to be regarded as the infantile form of myxœdema, and, like that disease, is due either to the congenital absence of the thyroid body, or an absence of its functions. Formerly it was assumed to be a form of goitre; but an enlarged thyroid has rarely been associated with cretinism, and the cases cited have not been fully investigated, as the thyroids were not examined post mortem. It is endemic in certain parts of Central Europe, but the sporadic form is met with in Great Britain, and is not related to locality.

Symptoms and Signs.—The condition as regards the subcutaneous œdema, supraclavicular swellings, and mental defects is much the same as in myxœdema, only more marked. No

disease presents more strikingly characteristic features. The child is dwarfed or stunted in growth; the face is very ugly, moon-shaped, and the cheeks hang in pendulous folds; the tongue is too large for the mouth; and the voice is peculiarly harsh or squeaky, like Punch in the show. The hair is abundant but coarse, except over the swellings between the scapulæ, which are covered by a soft down-like hair. The swellings are due to fatty deposits. The belly is very prominent and pendulous; umbilical and inguinal hernia are very common. The gait is clumsy, and of a waddling type. The sexual organs are rarely developed, though in female children the menses may appear once or twice; but sexual desire is never excited, even in those cases that live to adult age. Cretins are usually incapable of being taught reading or writing, and their vocabulary is always limited. They are often peevish and cross, but the bulk are placid and affectionate. Death most often occurs during childhood, though a few cases have reached adult life, without, however, advancing at all in intelligence.

Treatment.—Nutritious diet, rich in animal food. Removal from the place where the disease appeared, to a more favourable climate. Frequent baths and systematic exercise of the muscles. In very young children *Liq. Thyroidei* mij, or *Thyroideum Siccum* gr. j, once daily. In older children five grains of the extract in tabloid form once daily. The younger the child the greater the improvement. Treatment must be continued throughout life.

Ætiology.—1. Hereditary predisposition, the ancestors of cretinic children having, in some cases, suffered from goitre, and thereby possibly transmitted a weakness in the development of the thyroid body in successive generations.

2. Unhealthy dwellings and faulty diet.

3. Climatic and geological influences. Cretinism is very common in deep valleys, especially among limestone formations, where the air is humid, and there is but little sunlight.

4. Intemperance and insanity in the parents, and inherited mental disease.

5. The proximate cause is of course atrophy or absence of the thyroid gland.

GOITRE.

A disease due to parenchymatous enlargement, adenoma, or cystic disease of the thyroid gland. It is endemic in certain parts of England, especially Derbyshire ("Derbyshire neck"), and in many parts of Europe. It is most common among limestone formations, affects women chiefly and begins in early adult life. Goitrous parents tend to produce cretinoid children.

Symptoms.—Fulness in the neck; thyroidal tumour moving with deglutition, the enlargement being either uniform or irregular as the whole thyroid or only a part of it is affected; and evidence of pressure on trachea, œsophagus, sympathetic, or recurrent laryngeal nerve. Sometimes there may be paroxysmal dyspnoea, which may prove fatal.

Treatment.—Thyroid extract (Thyroideum Siccum gr. ij t.i.d. continued in decreasing doses for some months), or iodide of potassium if the thyroid does not agree. Paint with Lin. Iodi. Treat cysts or adenomata surgically. Boil the drinking water.

II. DISEASES OF THE SUPRARENAL GLANDS.

ADDISON'S DISEASE

Is a peculiar constitutional affection, first described in 1855, by Dr. Addison of Guy's Hospital, as a "disease of the suprarenal capsules, attended with a bronze discoloration of the skin, and incurable progressive anæmia." His description, however, requires modification; as *anæmia* is by no means always a prominent feature. Dr. Byrom Bramwell sums up the condition in his beautiful Atlas (p. 51), as a condition marked by—

1. *Asthenia*, feebleness of the action of the heart, and the symptoms and signs which result therefrom.

2. *Nausea*, vomiting, and other symptoms which result from gastro-intestinal irritation.

3. *Pain* in the neighbourhood of the suprarenal bodies.
4. *Pigmentation* of the skin and mucous membranes.
5. *Anæmia*.—Often absent until the later stages.
6. Symptoms due to derangements of the cerebro-spinal nerve apparatus, such as headache, delirium, convulsions, etc.
7. The *absence* of—
 - (1) Elevation of temperature.
 - (2) Marked emaciation.
 - (3) Signs or symptoms of local organic disease (other than the suprarenal capsules) to account for the asthenia and other symptoms enumerated.

Ætiology. The disease occurs twice as often in men as in women. In 80 per cent of the cases the lesion of the capsules is tubercular. In most of them tuberculous lesions are also present elsewhere.

Pathology.—At present is obscure. All observers agree that it is essentially a disease of the adrenal bodies, plus implication of the abdominal sympathetic system. Some lay most stress upon the adrenal disease; others upon the involvement of the sympathetic system. Bramwell, after a most exhaustive analysis of this disease, considers it "*to be, in the majority of cases, a tubercular degeneration of the capsules primarily, and that the alterations in the nervous structures are secondary.*" The lesions of the sympathetic nerves are not constant.

So far so good; but when we attempt to explain the symptoms we are at once met by the fact that the functions of the abdominal sympathetic, and also of the adrenal bodies, are still imperfectly understood.

It is held, judging from the enormous vascular supply of the adrenal bodies, that they are blood glands, and that their function is to prevent the retention within the system of certain poisonous products, which, if retained, produce the peculiar symptoms of Addison's disease; and if we bear in mind the case of thyroid myxœdema, this sounds feasible. It is at all events the most generally accepted theory.

If it be due to the involvement of the sympathetic, the symptoms must be the outcome of trophic changes, and the

consequent disturbed and imperfect metabolism through imperfect innervation.

Anatomical Changes are—

1. Atrophy of one or both glands ; presenting
2. The various stages of tubercular degeneration—
 - (1) Interstitial growth.
 - (2) Fatty degenerated patches.
 - (3) Caseous masses.

The semilunar ganglia are degenerated and much pigmented, and they may through cicatricial contraction become entangled as it were in the diseased tissue of the adrenals. The nerve fibres show extensive sclerotic changes.

Drs. Alezais and Arnaud assert that "Addison's disease will not result if the *pericapsular ganglia* be not affected, though the other portions of the adrenal bodies are at the same time extensively diseased."

We have already summed up the main symptoms. It is peculiar that even when the exhaustion and anæmia are profound, the temperature tends to keep down ; this is in marked contrast to pernicious anæmia.

The pigmentation is most marked where pigment is normally found—*i.e.*, areolæ, scrotum, axilla, etc. The mucous membranes are often deeply pigmented, presenting blackish patches. The marked exhaustion is altogether out of proportion to the general condition, and it must not be forgotten that the slightest untoward circumstance, such as a simple purge or chill, may cause death. Provided, however, the patient be treated as a hothouse plant, the disease may run a protracted course. In some cases, after very slight symptoms (and a still slighter amount of bronzing) have been noticed for a few months, the patient, without any obvious cause, sinks into a semi-comatose state which rapidly proves fatal.

Diagnosis.—It must be remembered that bronzing may occur in pregnancy, through exposure to the sun, through dirt and vermin, in diabetes, etc.

Treatment can be summed up as follows :—Treat the patient

with the extremest precautions against cold, worry, or fatigue. Complete rest in bed may be necessary. It has been suggested to introduce the extract, etc., of healthy adrenal bodies, after the manner of the thyroid in myxœdema. If given by the mouth, a quantity equal to 15 grains of the gland should be given twice daily, increasing gradually to 5i twice daily. If given hypodermically one-third of the above dose should be used. The results are not as good as those in myxœdema, improvement being usually temporary. Arsenic, phosphorus, iron, strychnine, etc., have their advocates.

III. DISEASES OF THE SPLEEN.

The diseases of the spleen are for the most part secondary. Thus one may meet with acute congestion in acute infections; passive congestion in cardiac disease or engorgement of the portal circulation; infarction, usually anæmic, but sometimes hæmorrhagic, associated with perisplenitis and consequent splenic friction; abscess in septic conditions; amyloid disease, in which the organ is very greatly enlarged; and tertiary syphilitic conditions. The characters of the spleen in leukæmia will be referred to under that disease.

MOVABLE SPLEEN

Is frequently associated with general enteroptosis. The organ is usually somewhat enlarged, and may descend into the abdomen so as to be grasped by the hand and moved about. The splenic notch may be recognised. Slight pain in the left side of the abdomen is the chief symptom. A belt and carefully adjusted pad may serve to retain the spleen in its normal position, or no treatment may be required.

SPLENIC ANÆMIA.

A chronic anæmia associated with enlargement of the spleen and tendency to gastric or other hæmorrhages. It occurs chiefly in males.

Pathology.—The spleen is much enlarged, its fibrous tissue being increased, and the endothelium of the blood sinuses proliferating, while the splenic pulp is atrophied. Perisplenitis and infarcts are also present. The liver is sometimes cirrhotic, and jaundice or ascites may occur. Anæmia may be a comparatively late symptom. The red corpuscles may fall to 3,000,000 per cmm., the hæmoglobin being still more deficient. The leucocytes also are *reduced* in number. The splenic origin of the disease is indicated by the fact that removal of the spleen has in some cases resulted in cure.

Symptoms.—The patient may first be troubled by digestive symptoms, or he may note the presence of a “lump” in the left side of the abdomen. The enlargement of the spleen may be very great, *but the lymphatic glands are not enlarged*. Splenic friction may be heard, due to perisplenitis. Progressive anæmia sets in. Severe hæmatemesis, followed by melæna, is common. Other hæmorrhages may occur, as from the nose, gums, or kidneys. Ascites and jaundice may characterise the late stages.

Treatment.—Open air, *arsenic*, and iron. Splenectomy is of little use in the late stages, if the strength is already much exhausted, but in the earlier stages it has led to cure.

CHARACTERS OF A SPLENIC TUMOUR.

1. The enlarged spleen extends *obliquely* downwards and forwards into the abdominal cavity from the left hypochondriac region. It may reach downwards to the iliac crest, and may reach to the umbilicus, or even farther, to the right.
2. The fingers cannot be pushed under the ribs in the left hypochondrium.
3. The surface is usually firm and smooth, and the edges rounded. If the enlargement is sufficient, the splenic notch may be felt in the epigastric region, to the left of the middle line.

4. Dulness is uninterrupted over the area occupied by the tumour, and is continued upwards into the normal area of splenic dulness. In tumour of the left kidney, the dulness is broken by a band of tympanitic percussion, due to the position of the descending colon in front of the kidney.
5. Posteriorly the lower border of splenic dulness meets the dulness of the spinal muscles at an acute angle. In renal tumour the whole region of the loin is dull to percussion.

If, on percussing along a line drawn from the umbilicus to the apex of the left axilla, the note is found uniformly tympanitic over the abdomen, and uniformly clear over the lung, there can be no important enlargement of the spleen.

IV. DISEASES OF THE PITUITARY BODY.

ACROMEGALY

Is a peculiar disease characterised clinically by an excessive growth chiefly of the face and extremities, and associated pathologically with hypertrophy of the pituitary body. In some cases the thyroid gland has been found enlarged, and a persistence of the thymus gland noted, but these two changes are looked upon as accidental occurrences.

Symptoms.—Rarely is a more characteristic clinical picture presented than a confirmed case of acromegaly. The face is egg-shaped, with its broad end downwards; the lower jaw-bone particularly is much increased in size, and may cause the lower teeth, which are wide apart, to project, giving anything but a pleasing appearance; the tongue is somewhat enlarged and may cause a similar leathery speech to that of myxœdema. The extremities are markedly altered, the hands being huge, spade-shaped, and the nails broad and large. The lower extremities show similar abnormal overgrowth, and may render the gait peculiarly clumsy. The overgrowth does not extend beyond the wrists and ankles, the long bones being

but slightly affected. The spine is often of the kyphotic type, and the abdomen being thrown outwards at the same time causes a characteristic "double hump." There is much general lassitude and weakness, and usually *marked headache* and *anæmia*.

The menses in women may be arrested. Defects of vision are often present, the more common being either single or double hemianopia; such conditions, however, are not always permanent. Polyuria and glycosuria may be present in some cases. The skin often presents pedunculated tumours, and in marked contrast to myxœdema often is bathed in sweat.

Pathology.—At present is very obscure, though, from the almost constant presence in these cases of a much hypertrophied pituitary body the condition seems to be dependent on changes in that gland. This, however, must be regarded at present as purely speculative, for cases of *atrophy* of the pituitary body have apparently been attended with no particular changes. The persistence of the thymus gland rests upon the slender evidence of increased dulness in its normal area.

Treatment.—Arsenic, strychnine, massage, galvanism, and the injection of extract of thyroid gland, or extract of pituitary body, may be tried. None of these forms of treatment seems to affect the progress of the disease.

EXAMINATION OF THE BLOOD.

In cases where anæmia or other altered conditions of the blood may be suspected, the diagnosis should be confirmed by an examination of the blood itself. In such an examination several points must be considered. They are as follows:—

1. Estimation of the number of corpuscles, red and white.
2. Estimation of the percentage of hæmoglobin.
3. Observation of the changes in form or size of the red corpuscles.
4. In cases where the white corpuscles are increased in number, determination of the *kind* of corpuscle which is principally increased.

5. Presence of micro-organisms or other parasites in the blood (less important from the point of view of blood diseases, but often confirmatory of the diagnosis of acute infections, such as anthrax, enteric, tubercle, etc.).
6. Presence of other abnormal constituents, as pigment (melanæmia) free in the blood plasma (severe malarial cachexia, melanotic sarcoma, etc.), or fat (lipæmia) in cases of diabetes mellitus.

1. and 2. The number of corpuscles and the percentage of hæmoglobin may be estimated, for the first, by the hæmocytometer of Gower or Thoma-Zeiss, and for the second by the hæmoglobinometer of Gower or Oliver. For details consult a recent manual of clinical medicine.

The blood should be taken from the finger-tip, or the lobe of the ear, after careful cleansing of the part. The finger should not be compressed.

It will be found upon examination that the various anæmias differ in the relative proportion of hæmoglobin and red blood corpuscles. In secondary anæmia, for example (following phthisis, cancer, syphilis, etc.), these two constituents are diminished in much the same proportion. In chlorosis the red cells are slightly defective, the hæmoglobin much more so. In pernicious anæmia there is an enormous destruction of red corpuscles, while the percentage of hæmoglobin is usually somewhat higher.

3. The red corpuscles may be altered in shape and size. Alterations in shape (poikilocytosis) are common to all forms of anæmia, but most profound in the graver forms. Alterations in size may result in smaller corpuscles (microcytosis), or in larger (macrocytosis). The more frequent are such alterations, the more likely is a diagnosis of pernicious anæmia. And, especially in pernicious anæmia, one may find *nucleated* red corpuscles, either of ordinary size (normoblasts), or much larger than the normal (megaloblasts).

4. Increase in the number of white corpuscles (leucocytosis). This may be—

- (1) Physiological, as after meals, or

- (2) The accompaniment of inflammatory diseases and specific fevers ; *but not enteric*, unless complications have arisen.
- (3) Leucocytosis is an essential feature of leukæmia or leucocythæmia, by whichever name it may be called. It is sometimes possible to determine the form of leukæmia which is present by observation of the kind of leucocyte principally increased. In lymphatic leukæmia the lymphocytes are present in enormous numbers, in splenomedullary leukæmia the polymorphonuclear and eosinophil leucocytes. This distinction, however, does not hold universally.

The alterations referred to under (5) and (6) do not require separate discussion here.

Opsonins.—If in a capillary pipette there be mixed equal volumes of leucocytes washed free of blood plasma, and of an emulsion of a pathogenic micro-organism, and if the pipette be then incubated at 36° C. for fifteen minutes, it will be found that no phagocytosis has occurred. If to the former ingredients an equal quantity of healthy blood serum be added, and the same process carried out, a greater or less degree of phagocytosis will be found to have taken place, showing that the serum contains substances essential for phagocytosis. These substances are known as *opsonins*. Their relative amount in any individual for a particular organism is measured by (a) counting the number of bacteria ingested by a definite number of leucocytes mixed with the serum of that individual, and (b) comparing this with the number ingested when normal serum is used. The ratio between the patient's phagocyte count and the normal phagocyte count is the *opsonic index*. The introduction of a vaccine (dead micro-organisms sterilised by heat) into the blood is followed first by a temporary lowering (*negative phase*), then by a more lasting raising (*positive phase*), of the opsonic index. The raised level persists from ten days to a fortnight or more (*high tide of immunity*), then gradually declines to the normal level. If a large dose of the vaccine be

given the negative phase may be prolonged, and if during this stage the dose be repeated, the effect is cumulative. Malaise and pyrexia develop, and the patient's resistance may be dangerously lowered. A cumulative effect is not apparent in the positive phase.

In health, for each individual, the opsonic index does not vary; but it may in different individuals remain either slightly below or slightly above the normal, varying from 0·8 to 1·2. In disease, the index is low in localised infections, and either high or fluctuating in systemic infections. One individual may have a high index for one organism and a low one for another, *i.e.*, the opsonins are specific. The low index of localised infections precedes and favours the onset of the infection. It continues low because the organisms remain local, and as they do not enter the blood no positive phase or period of immunity is produced. It is high or fluctuating in systemic infections because the organisms enter the blood and so produce a series of auto-inoculations with negative and positive phase. The doses, however, are sometimes excessive, or the intervals between them too brief, so that the dangerous effects of a prolonged negative phase are obtained; but in other cases the repeated auto-inoculation leads to successful immunisation, and hence to recovery.

Treatment by vaccines is based upon these considerations, the object being to raise the opsonic content of the patient's blood against the organism of the particular disease. The inoculations must always be controlled by estimation of the opsonic index and must not be repeated during the negative phase. The method has so far been applied chiefly in localised staphylococcic infections (acne, furunculosis), and in localised tuberculous disease. In such cases it has proved strikingly successful.

ANÆMIA.

Anæmia really means want of blood, but the term is employed in a much wider sense, and under anæmia are included—

1. Oligæmia—deficiency of blood.
2. Hydræmia—thin or watery blood.
3. Oligocythæmia—a diminution of the number of corpuscles.
4. Poikilocythæmia—irregularity in the shape of the corpuscles.
5. Oligochromæmia—deficiency of blood-colouring matter.

Special Anæmias.—

- (1) Symptomatic or Secondary.
- (2) Chlorosis.
- (3) Pernicious Anæmia.

It will save much recapitulation, and help to avoid much haziness regarding these affections if we start with a clear idea what anæmia really means, as regards its effects upon the body generally.

Pathology.—We must expect imperfect oxygenation, deficient nutrition, and, therefore, impaired function, fatty degeneration, atrophy of certain tissues, and softening of the blood-vessels; a vicious cycle of retrograde changes thus begins, each new failure on the part of an organ increasing the original condition. The nerve centres are frequently hyperexcited at first, as shown by powerful emotional manifestations; or there may be diminished sensibility, as seen in the dilated pupils, which show the depressed condition of the retina. Œdema in unsupported tissues is most common. Owing to muscular weakness dilatation of the heart is not infrequent. Hæmorrhages from any of the mucous surfaces, and especially retinal hæmorrhages, are frequent in the graver anæmias.

SYMPTOMATIC OR SECONDARY ANÆMIA.

Causes.—

Deficiency of food, either through actual want, or inability to take food—as in cancer of the œsophagus, etc.

Excessive discharges—i.e., pyæmic abscesses, diarrhœa, etc.

Cachectic conditions—phthisis, cancer, syphilis, etc.

Hæmorrhages.—The blood in these cases, if the bleeding is

profuse, becomes rapidly diluted with lymph. There is also leucocytosis.

Certain occupations—lead workers, colliers, etc.

In these cases removal of the cause, when that is possible, and placing the patient under favourable hygienic conditions, bring about a cure.

CHLOROSIS.

The usual definition in text-books is,—a peculiar anæmia attended with a greenish, transparent, wax-like condition of the skin, common in females between the ages of fifteen and twenty-one.

Symptoms.—Languor, dyspnœa, palpitation on exertion, headache, noises in the ears, and dyspeptic symptoms, as in symptomatic anæmia. The more characteristic symptoms in addition are plumpness; systolic murmurs at the base of the heart; “*bruit de diable*” at the lower end of the jugular vein; constipation; irregular, profuse, or scanty menstruation, and the waxy appearance of the skin already referred to. Mitral regurgitation is sometimes present,—the result of relative insufficiency of the valve, following upon dilatation.

Pathology.—The red corpuscles are somewhat diminished in number and of various shapes; some swollen, others crenated, but all *show a remarkable diminution of hæmoglobin*, which must be considered the chief peculiarity of chlorosis. There cannot be the least possible doubt that chlorosis is due to defective blood formation—something wrong in the building up, *not an excessive breaking down*. (According to Lorraine Smith, the amount of blood plasma is greatly increased, and the hæmoglobin is therefore only *relatively* diminished.) The theories advanced to explain such a condition are numerous, and the following may be borne in mind for examination purposes:—

1. Due to constipation. (Sir ANDREW CLARK.)
2. That the period of puberty causes an increased demand on the blood-forming glands, which are unable to meet the call.

3. The demands of puberty reveal a congenital narrowness of the aorta, which prevents a proper supply of blood to the various tissues.

4. *That the organic iron compounds of the food are broken up by excessive decomposition in the alimentary canal, and the iron thus liberated, is rendered incapable of being absorbed into the blood.* (BUNGE.)

5. Excessive loss of blood at the period of puberty (menorrhagia), leading to dyspeptic conditions, anorexia, and defective absorption of iron. (STOCKMAN.)

It is painfully interesting to note that none of the above explains the disease, for constipated subjects are not always chlorotic, chlorosis is not confined entirely to women, and chlorosis may be present with a sound aorta. At present we may assume that chlorosis is due to many factors,—the chief being the *adolescent period*, which causes an increased demand on the blood-forming glands, and develops latent weaknesses; further, such period conduces towards an unstable state of the nervous system, manifested by dyspepsia, constipation, etc.

As to the *immediate* cause, it is highly probable that Bunge's or Stockman's theory is correct.

Treatment.—Is highly satisfactory. Fresh air, gentle exercise, nitrogenous food, purgatives, and iron will cure the worst cases.

An excellent combination is iron, aloes, and carbonate of potash; but Bland's pill, especially in the bipalatinoid form, is the most generally useful. If there be dyspepsia, ferric salts should never be given, but the non-irritating ferrous salts should be used instead. The iron is sometimes advantageously combined with arsenic. Manganese, often recommended, is useless. The iron acts in two ways:—

1. It combines with many of the products of decomposition in the duodenum, and thereby forms insoluble and inert salts, which are excreted, thus preventing the pernicious gases, etc., entering the portal system.

2. A small amount of the iron itself *is absorbed and assimilated*.

Carbohydrates as a rule are harmful. Chlorotic girls should be released from all studies, and exciting literature should be kept from them.

PERNICIOUS ANÆMIA.

This is a progressive and profound anæmia, developing without any evident cause, and ending most frequently in death.

Ætiology.—Affects males somewhat oftener than females. Is rare under twenty-five years of age. Some cases have started during pregnancy. The disease, according to Hunter, is a chronic febrile infection, due to auto-intoxication from the gastro-intestinal tract. It must, however, be remembered that cases presenting all the clinical appearances have been found, *post mortem*, to be due to cancer of the stomach or bones, anchylostomiasis, etc.

Pathology.—*The Blood* shows a remarkable diminution of red corpuscles; they may sink to less than half a million per cubic millimeter. The amount of hæmoglobin in each corpuscle, so far from being diminished, is often relatively increased. The corpuscles exhibit a number of various shapes, some are tailed, others crenated; often the hæmoglobin can be seen protruding prior to its escape from the stroma; some discs are extremely minute, and may be nucleated. Microcytes, macrocytes, normoblasts, and megaloblasts, may all be found. *The blood when shed coagulates with difficulty.*

The Liver shows marked changes—

1. Is exceedingly rich in iron.
2. There is an excess of pigment within the liver-cells.
3. There is fatty degeneration in the central third of each lobule.

The *spleen, kidney, and bone-marrow* also show excess of iron pigment. Punctiform hæmorrhages may be found in all the organs.

There is but little doubt that pernicious anæmia is due to *excessive destruction of the cellular elements of the blood.*

Hunter maintains that pernicious anæmia is due to an organism causing hæmolytic. The disease is very constantly associated with oral or gastric sepsis, and in these conditions is found a permanent nidus for the growth of the microbic poison.

Prognosis.—Highly unfavourable, most cases die in from six to fifteen months.

Symptoms.—The early symptoms are: “(1) Glossitic, gastric, and intestinal; accompanied by (2) Hæmolytic symptoms—lemon colour, biliousness, with or without jaundice; (3) Febrile and nervous disturbances; (4) a remarkable degree of anæmia, out of all proportion to the severity of any symptoms present” (Hunter). As the case progresses these symptoms recur with greater severity, but with periodic remissions at intervals of three or four weeks. Hæmorrhages are frequent, especially retinal hæmorrhages and hæmoglobinuria or hæmaturia. Epistaxis or melæna may also occur.

Though of course the muscular wasting is considerable, the subcutaneous fat is often increased; hence the wasting is not apparent, and the patient may present a fairly well-nourished appearance as regards the general contour of the body.

Treatment.—Tonics of all kinds may be used to meet the symptoms of the individual case. The only hæmatinic of any value is *arsenic*, which must be given persistently, and gradually pushed to large doses. The septic conditions of the mouth must be rigorously treated, and all carious teeth removed. For gastric antisepsis, after lavage of the stomach, salicylic acid should be administered, and for intestinal antisepsis, small doses of mercurial preparations.

In cases where the infection is “firmly rooted in the mucosa,” the poison must be attacked in the blood. For this purpose Hunter uses injections of antistreptococcic serum, beginning with small doses (5 cc.) repeated after a few days’ interval. By these means he has been able to prolong life and to maintain health for over seven years. Treatment must be resumed on the earliest appearance of a relapse.

LEUKÆMIA OR LEUCOCYTHÆMIA.

Leukæmia is an affection characterised by persistent increase of white corpuscles, hæmorrhages, and changes in the blood, marrow, spleen, lymphatic glands, etc.

Ætiology.—The disease may develop at any age, but by far the greater bulk of cases occur in the middle-aged. Men are more often attacked than women. The more important ætiological factors that have been observed are—

Heredity,
Syphilis,
Malaria,
Traumatism, and
Pregnancy.

All these are common causes of disease, and leukæmia is comparatively rare. They do not really explain its origin.

Pathology.—*The blood* is markedly altered, and shows—

1. The white corpuscles to be enormously increased, so that the proportion of white to red corpuscles may be as high as 1 to 6, or even 1 to 3, instead of 1 to 400 or 500 as in normal blood. The white corpuscles are of different shapes and sizes, and hence an attempt has been made to distinguish various types of the disease, lymphocytes, as has been already mentioned, being derived from the lymphatic glands, while a preponderance of polymorphonuclear and eosinophil cells is held to point to splenomedullary leukæmia. Thus the preponderance of any particular shape points to the structure mostly involved. The red cells are diminished in number: normoblasts and even megaloblasts are found.

2. An excess of fat, so that a drop of the leukæmic blood dropped on to white paper leaves a greasy stain.

3. The presence of elongated, octahedral, colourless, phosphatic crystals (similar to those found in asthma).

4. The abundance of hypoxanthin and other antecedents of urea.

The Spleen.—It is usually firm, much enlarged (in the

splenomedullary form it may weigh as much as 15 lbs., but in the lymphatic form the enlargement is less notable), and may be bound by adhesions to the abdominal wall, the diaphragm, or the stomach. The capsule is much thickened. On section, the organ presents a pinkish appearance, appears fatty, and feels greasy to the touch. The pulp is dark, and the Malpighian tufts stand out as pale spots. The sinuses are often distended with leucocytes. Hæmorrhagic infarctions, old and recent, are very common. Later, the changes are more fibrous in character, and the splenic pulp may be much atrophied from the pressure.

The Lymphatic Glands.—Nearly all the lymphatic glands are enlarged and softened; later, they may assume the same characters as observed in Hodgkin's disease.

The Marrow.—The marrow may be dark brown in colour, or present a peculiar yellowish-red appearance. Large numbers of nucleated red corpuscles and non-nucleated leucocytes are found.

The other tissues and organs, such as the liver, heart, etc., show those grave changes consequent upon deficient oxygenation, *plus* extravasations of blood. The liver is also materially enlarged, and its capillaries contain great numbers of leucocytes. Metastatic leukæmic changes are found in the liver and kidneys.

Symptoms.—The onset is most insidious, and the patient usually applies for medical advice on account of breathlessness, dyspeptic symptoms, palpitation, and other symptoms of anæmia. Hæmorrhages of various kinds may be the first symptoms. When the disease has advanced, the condition of the patient is most characteristic. The abdomen is prominent; the countenance of a deadly white, or with a slightly sallow tint; the sclerotic of a pearly lustre; pupils dilated, mucous membranes blanched. The pulse is soft and compressible, contrasting well with the high-tension pulse of chronic Bright's disease. Hæmorrhages are common under the skin, in mucous membranes, and into the retina. The retinal changes are often most marked, the vessels being tortuous and often distended with the white corpuscles. Towards the end, grave changes

set in and intermittent diarrhœa, *attacks of fever*, and œdema or general dropsy usher in a fatal issue. It must not be forgotten that the urine usually is of high specific gravity, containing excess of uric acid and urea. The pallor, usually so well marked, is sometimes wanting. The special points to note are the condition of the blood, marked exhaustion, pyrexia, and the tendency to dropsy associated with highly coloured urine.

Characters of the enlarged Spleen.—It enlarges in the axis of the tenth rib, and, therefore, tends to go forward to the right iliac fossa. If much enlarged, it is found to be present as a firm, hard tumour, having a distinct notch in its anterior border; it may reach from the flank down to or below the umbilicus, and weigh from six to fifteen pounds. Note in contrast to floating kidney—the colon never lies in front of an enlarged spleen.

Prognosis is always bad, death occurring in from one to two years.

Treatment.—Everything must be done to protect the patient from cold, and the general hygiene must be as perfect as possible. Massage and galvanism may be applied to the spleen, but do not effect much good. The medicinal treatment is as described under the treatment of Pernicious Anæmia.

HODGKIN'S DISEASE OR LYMPHADENOMA.

It is an affection characterised by progressive enlargement of the lymphatic glands, destruction of red corpuscles, and secondary lymphoid growths in the various organs.

Ætiology.—It may attack any age, but is by far more common in children and young adults. No special ætiological factor is known. Possibly syphilis, tubercle, and traumatism are important factors, inasmuch as they weaken the glandular system.

Pathology.—The exact cause of Hodgkin's disease is quite unknown, though there can be no doubt that it is due to some

irritant formed either in the blood, or introduced from without, but which, when in the blood, causes an overgrowth of lymphoid tissue.

The affected glands are usually much enlarged, single, painless, and non-adherent to the skin. Though they may feel to the touch hard and solid, they are far more frequently somewhat soft and elastic. *Their two great characteristics are—they tend neither to suppurate nor to caseate.* Of course, like all other growths, they may under certain circumstances suppurate and mass together; but, as Dr. Bramwell emphasises, “such results are to be regarded as accidental.” The disease may cause large deposits of lymphoid tissue in the subcutaneous tissue and marrow of long bones.

Histologically.—

The Glands.—The chief change is a general hyperplasia of the whole gland, with thickening and increased firmness of the capsule, accompanied by the formation of a large number of small cells resembling leucocytes. The amount of new fibrous tissue formed governs the degree of hardness: if the lymphoid cells are abundant the gland is soft; but when the fibrous tissue element is in excess the glands are hard.

The Spleen.—Is always enlarged in Hodgkin's disease, but not nearly to the same extent as in leucocythæmia. In consistence it is firm, and purple patches from venous congestion often appear on the surface. On section can be seen golden-yellowish masses, consisting of lymph corpuscles and pigment enclosed in a fibrous reticulum. The Malpighian bodies appear as translucent yellowish suet-like masses. The general fibrous stroma is much increased, as in the lymphatic glands. Lymphoid tissue is sometimes found in the bone-marrow, and there may be lymphoid nodules in the liver.

Symptoms.—Briefly are—

1. Enlargement of the lymphatic glands.
2. Anæmia—often marked decrease of red corpuscles, and a normal or *slightly* increased number of leucocytes.
3. Symptoms pointing to the virulent nature of the disease

—*i.e.*, marked emaciation, cachexia, secondary deposits of lymphoid tissue.

4. Attacks of pyrexia.

5. Symptoms due to pressure from the lymphoid growths.

The glandular enlargement most frequently begins in the anterior chain of glands at the posterior border of the sternomastoid, then the axillary, inguinal, and finally all the glands of the body may show extensive changes. Often both sides of the neck become involved, and the growths meeting anteriorly in the middle line may compress the trachea to a dangerous extent. Osler points out that when the abdominal glands are affected the sympathetic system is often profoundly disturbed, and pigmentation of the skin may occur. The enlarged mediastinal growth may cause severe dyspnoea, inequality of the pupils, pleurisy, etc.

The periodical elevations of temperature may be so marked as to simulate ague. It will be at once seen that almost any symptom may crop up in Hodgkin's disease, and no wonder if we remember the extensive nature of the disease, and the important structures that may be involved thereby. Lastly, note that in some cases the red corpuscles are not diminished until marked cachexia has set in.

Treatment.—It has been suggested that when the disease has affected only one set of glands, the diseased portion should be excised; such procedure is certainly as rational as operative measures in other malignant conditions. We may hope for benefit from subcutaneous injections of arsenic, combined with massage and careful hygiene. The disease is, however, only too frequently fatal in from two to four years.

SCURVY

Is a constitutional affection characterised by great debility, a spongy condition of the gums, a tendency to hæmorrhages, and cachexia.

Ætiology.—Is much less common now than in former times.

The disease is usually associated with improper and insufficient food, unhealthy hygienic surroundings, etc. Sailors afford the best examples, as a rule, of this disease.

Pathology.—There are three theories as to the causation of scurvy—viz., that it

1. Depends on the presence of a specific organism. No such organism has yet been found.

2. Deficiency of *potassium* salts in the blood.

3. Reduction of the alkalinity of the blood, not necessarily through the absence of potash, but a reduction in quantity of all the salts that tend to keep the blood alkaline.

Moreover, as citrates, malates, tartrates, etc., become changed into carbonates in the blood, we may assume at present that scurvy is really due to an inadequate supply of organic acids, salts, etc., which are to be found in fresh vegetables, fruits, etc.

Post mortem wide-spread hæmorrhages are found. The blood has the characters of secondary anæmia.

Symptoms.—Are insidious at first; the patient becomes weak, breathless, drowsy or languid, with more or less aching of the bones and joints generally. The gums become soft and swollen, bleeding easily on the slightest pressure.

As the disease becomes marked, the teeth may come out, the mouth becomes ulcerated and emits a frightfully foetid odour. Petechial hæmorrhages around the hair follicles may be followed by large subcutaneous extravasations of blood on the extensor aspects of the limbs, into the conjunctivæ, popliteal spaces, etc. The patient assumes a cachectic appearance, and is rendered quite unfit for mental or physical exertion. In some cases a peculiar form of night blindness develops; this condition is not dependent on permanent ocular changes, though optic atrophy has been observed in more than one case.

Treatment.—

1. General hygiene must be attended to.

2. Diet should consist of good soup, fresh milk, cream, etc. Effervescing drinks made with fresh lemon juice are of special

importance. Fresh fruits and vegetables should be liberally given.

3. Medicinal.—Quinine dissolved in citric acid. Local measures for the bleeding gums, nitrate of silver, antiseptic mouth-washes, etc.

HÆMOPHILIA

Is a peculiar diathesis characterised by a tendency to excessive and uncontrollable bleeding. It differs from purpura in being always present throughout the patient's life.

Many varieties have been described such as—

1. Those in which a slight traumatism is followed by excessive hæmorrhage.

2. Those which exhibit the condition after injuries of certain regions only.

3. Those in which the hæmorrhages take the form of attacks of spontaneous bleeding from the nose, uterus, mucous membranes, etc.

Pathology.—Unknown, as no *constant* changes have been found *post mortem*, either in the blood-vessels or blood; probably the fault lies in some structural changes in the walls of the capillaries. The blood itself is much less easily coagulable than in normal individuals; leucocytes and blood platelets are often diminished in number. The diathesis is marked by being hereditary, and is transmitted through the female line, although the male is most liable to the *disease*.

Symptoms.—Are sufficiently obvious, and need no detailed description. It must be remembered that in these cases death has followed the extraction of a tooth, cutting a corn too deeply, snipping a wart off, and other usually trifling operations. Often the first severe attack of bleeding occurs in infancy, or, on the other hand, it may first appear after adult age.

Treatment.—When we suspect the presence of this diathesis, a careful watch should be made over the patient, and on the

slightest appearance of hæmorrhage, energetic treatment with styptics must be at once employed. For hæmorrhages from the mouth or nose adrenalin or tincture of hamamelis (1 in 10) may be used, and for external wounds compression. Calcium chloride in doses of gr. xv every four hours is strongly recommended. Obviously, the bleeder must live on a non-stimulating diet, lead as even and placid a life as possible, avoid excesses of all kinds, especially the ingestion of liquids and other compounds which tend to raise the blood-pressure. No female of the hæmophilic stock should marry.

PURPURA.

Purpura is not a disease, but symptomatic of some grave change in either the blood or blood-vessels, whereby extravasations of blood into various tissues occur, producing red or blue patches which do not disappear on pressure or after death. Being symptomatic in character we may expect several varieties, such as—

1. A form associated with the malignant fevers—*i.e.*, those fevers in which the “rashes” become hæmorrhagic, as already described under typhus, black measles, etc.

2. A form associated with grave *constitutional* changes—*i.e.*, syphilis, chronic Bright’s disease, cardiac diseases, scurvy, etc.

3. A form associated with the circulation of certain poisons, to wit, snake poison, quinine, antipyrin, copaiba, mercury, etc.

4. A form probably neurotic in character, as seen in cases of myelitis, locomotor ataxia, severe neuralgia, purpura urticans, etc.

5. Purpura simplex, most often seen in children, unattended by fever or constitutional symptoms, and disappearing in a fortnight or less.

6. Purpura hæmorrhagica (*morbis maculosus Werlhofii*), a form which occurs without any apparent reason, and not associated with any particular condition. It is seen in two varieties:—

- (a) Non-infectious purpura hæmorrhagica (possibly an acute

form of hæmophilia). It attacks young subjects chiefly. The eruption is severe, the mucous membranes are also affected, and there may be renal or retinal hæmorrhages. Recovery may be speedy, or increasing anæmia may lead to death.

(b) Febrile purpura, associated with intense septicæmic infection, high fever, and a septic state of the mouth. Petechiæ, ecchymoses, and bullæ are seen, also hæmorrhages from the nose and gums. The issue is fatal.

7. Purpura rheumatica, or arthritic purpura, is as a rule not of rheumatic origin, although acute rheumatism may sometimes be complicated by purpuric rashes. To these the term peliosis rheumatica should be restricted. Arthritic purpura is accompanied by slight fever and pains in the larger joints, around which, upon the extensor surfaces, a rash appears. This is partly urticarial, and the purpuric lesions are seated upon the urticarial wheals. There may be œdema of the shins or even of the face.

Symptoms.—The presence of petechiæ in the skin, etc., is sufficiently obvious to demonstrate the condition. When not associated with any apparent cause the more common symptoms are—marked anæmia, sallow complexion, local œdema or general anasarca, and more or less severe muscular pains. Death may occur from progressive exhaustion, or from internal hæmorrhages. Unlike scurvy, the gums are not swollen, and there is no history of want of fresh air or vegetables, etc.

Treatment.—As we know nothing about the pathology of this disease the treatment must be entirely empirical, or experimental. Aim at removing any apparent cause of ill-health, restoring tone of blood-vessels, etc. Ergot, iron, turpentine, strychnine, gallic acid, opium, etc., may be tried. Arsenic is useful in purpura simplex, but should not be given if there is diarrhœa. Adrenalin internally, or calcium chloride gr. xv-xx every four hours are often useful. Rest in bed should be insisted on. In febrile purpura treatment is merely palliative.

DISEASES OF THE RESPIRATORY SYSTEM.

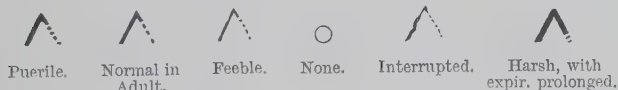
Dr. John Wyllie's Notes on Examination of the Respiratory System.

AUSCULTATION.

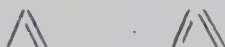
Listen for—(1) *Type of Breathing*, (2) *Accompaniments*, (3) *Vocal Resonance*.

1. TYPES OF BREATHING.

(1) VESICULAR or RUSHING—



Transition Type—



(Broncho-vesicular or "indeterminate" breathing.)

(2) BRONCHIAL or BLOWING—



(3) AMPHORIC—



NOTES.—(1) *Vesicular Breath-sounds*.—In the auscultation of normal Vesicular Breathing the *Inspiratory sound*, represented in the diagrams by a single line, is a fine, continuous, rushing sound, soft in the adult and loud in the child, and audible from beginning to end of the act. The *Expiratory*

sound, on the other hand, is thin in quality and of short duration, being audible only during the earlier part of the Expiratory act. In normal Vesicular Breathing the Inspiratory sound generally passes, as represented in the diagram, directly into the Expiratory without a break. Having, however, paid special attention to this point, Dr. W. believes that there is often, in perfectly normal Vesicular Breathing, a distinct break between the two sounds. When the breathing is quiet and easy, the Expiratory sound is often totally inaudible, even in children; but in such cases it can usually be brought out by causing the patient to breathe deeply. The term "prolonged expiration" is used to signify not a prolongation of the *act* of Expiration, but only a prolongation of the expiratory *sound*, resulting from the encroachment of the audible upon the inaudible part of the act. There is indeed one form of breathing, common to advanced Emphysema and Asthma, in which the act itself is really prolonged, being often much longer than the Inspiration. In such "Asthmatic" breathing, the type of respiration, primarily Vesicular, is as a rule totally masked by the loud wheezing accompaniments of both Inspiration and Expiration.

(2) *Bronchial or Tubular Breathing*.—The auscultatory sound of Bronchial Breathing, indicated in the diagrams by a double line, can be imitated, as pointed out by Skoda, by holding the tongue in the position for the pronunciation of the guttural *ch* sound (as in the German word *Ach*, or the Scotch word *Loch*), and causing the air to pass inwards and outwards over it. The blowing sound thus produced can be made to represent the various pitches indicated in the diagram. There is always in Bronchial Breathing a distinct break between the sounds of the Inspiration and Expiration, and the two sounds closely resemble each other. The higher-pitched varieties of Bronchial Breathing should be associated in the mind with conditions of consolidation of Lung Substance, such as that of Pneumonia, and the Low-pitched or Cavernous variety with Excavation, as in Phthisical cavity. *Bronchial Breathing is never produced by Bronchitis.*

(3) The *Amphoric* type of breath-sound can be well imitated by whistling with the mouth. Inspiratory and Expiratory sounds can thus be produced by causing the air to pass inwards and outwards, and the pitch can be varied according to the variety of Amphoric Breathing that is being imitated. Amphoric Breathing is best developed in Pneumothorax, but is also sometimes met with in *very large* Phthisical cavities.

(4) In the *Healthy Chest* the respiratory sounds are purely Vesicular (without harshness of quality or prolongation of expiration) over the whole surface of the lungs, except (*a*) opposite the *Roots of the Lungs*, at level of third dorsal vertebra behind, and lower part of manubrium sterni in front, where the proximity of the large Bronchi generally renders the breathing *Broncho-vesicular*, by the addition of a Blowing or Bronchial element, most distinct during expiration; (*b*) over the *Aper of the Right Lung*, especially above the Clavicle and Spine of Scapula, where, in health, from causes as yet

imperfectly ascertained, the Vesicular Breath-sound has very generally a more or less prolonged, and often harsh or even somewhat blowing, expiration.

The only example of purely *Bronchial* Breathing that can be heard on auscultating the healthy subject, is the "*Tracheal*" Breathing, to be obtained by placing the stethoscope over the Larynx or Trachea. This is low in pitch, and if heard over the apex of the Lung would be termed "Cavernous."

[Unfortunately some of the great original authorities on Auscultation applied the term "Bronchial" to the type of breathing heard over the roots of the Lungs, but, as this is partly of Bronchial and partly of Vesicular origin, the term "Broncho-vesicular" is much more appropriate.]

2. ACCOMPANIMENTS.

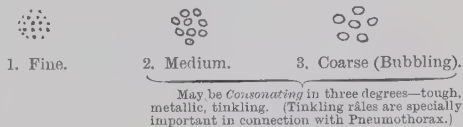
(1) FRICTION (in Pleurisy)—



(2) DRY SOUNDS, or RHONCHI (in Bronchitis)—



(3) MOIST RÂLES or CREPITATIONS (1 and 2 in Pneumonia ; 2 in Bronchitis ; 2 and 3 in Phthisis)—



3. VOCAL RESONANCE.

(1) SIMPLE INCREASE—

- (a) Slight, comparative.
- (b) Marked (Bronchophony).
- (c) Very marked (Pectoriloquy).

(The chief conditions which cause Increase of Vocal Resonance are Consolidation and Excavation of the Lung Substance.)

(2) SIMPLE DECREASE—

- (a) Slight, comparative.
- (b) Marked decrease.
- (c) Total absence.

(Decrease of Vocal Resonance is most frequently due to Thickening of the Pleura or to Pleuritic Effusion.)

(3) QUALITATIVE ALTERATIONS—

(a) *Ægophony* (Nasal timbre).(b) With metallic echo (*Amphoric Resonance* or *Nach-klang*).*(Ægophony occurs in Pleurisy with Effusion when the layer of fluid is thin. Metallic echo is one of the signs of Pneumothorax.)*

PERCUSSION.

1. HYPER-RESONANCE—

(1) Slight.

(2) Marked.

(3) Very marked (*Tympanitic note*).

(a) High-pitched.

(b) Medium-pitched.

(c) Low-pitched.

(Emphysema causes slight Hyper-Resonance. Relaxation of Lung substance, as in the Superior lobe when the Inferior is solid from Pneumonia or compressed by Pleuritic Effusion, is a cause of marked Hyper-resonance. Pneumothorax is the commonest cause of true Tympanicity.)

2. DEFICIENT RESONANCE—

(1) Slight, comparative dulness.

(2) Marked dulness.

(3) Absolute dulness.

*(Consolidation of Lung Substance, thickening of the Pleura, and Pleuritic Effusions are the chief causes of Deficient Resonance.)*3. MIXTURE OR DULNESS AND RESONANCE—*i.e.*, a Wooden or Boxy Note.*(This is one of the most important signs of a Phthisical Cavity.)*

4. SPECIAL QUALITY—

(1) Cracked-pot sound.

(Another very important sign of a Phthisical Cavity.)

(2) Bell sound, got with two coins and stethoscope.

(One of the signs of Pneumothorax.)

SUCCUSSION.

Called "Hippocratic"; used in Hydro- and Pyo-pneumothorax.

INSPECTION, PALPATION, AND MENSURATION.

1. FORM AND SIZE OF CHEST—

(1) Circumference of Chest at line of nipples.

(2) General Form (flat, barrel-shaped, etc.).

(3) Local alterations in Form (local flattening, bulging, etc.).

2. MOVEMENTS OF CHEST—

- (1) Number of respirations per minute.
- (2) General type of movement (thoracico-abdominal, abdominal, thoracic).
- (3) Rhythm and Volume of respirations; and their Special Character, as in the “Cheyne-Stokes” type, etc.
- (4) Local Movements (sucking in of intercostal spaces, etc.).
- (5) Deficient Expansion (over one apex, over one side, etc.).

3. PARTS OUTSIDE CHEST—

- (1) Box of Larynx (its upward and downward movement).
- (2) Alæ Nasi (their action in difficult breathing).
- (3) Bulging of Apices in neck on coughing.
- (4) Action of the Scaleni and other Extraordinary Muscles of Respiration.

4. PALPATE FOR THE VOCAL FREMITUS. (Note increase or diminution.)

EXTRA AUSCULTATION.

1. OF THE SOUNDS OF *Obstruction* IN RESPIRATORY PASSAGES.

- (1) OBSTRUCTION TO BREATHING { (a) From hardened or fluid Mucus. } Nasal Bubbling
IN NOSE { (b) From Paralysis of Alæ. } and Sniffing.
- (2) OBSTRUCTION IN BACK OF { (a) Nasal Snore. } Stertorous
THROAT { (b) Oral Snore. } Breathing.
- (3) OBSTRUCTION IN LARYNX { (a) Swelling of Cords. } Stridulous
{ (b) Paralysis or Spasm of Glottis. } Breathing.
- (4) OBSTRUCTION IN TRACHEA { (a) From Aneurism (Leopard-growl).
{ (b) Death Rattle.
- (5) OBSTRUCTION IN BRONCHI { (a) Musical Sounds (Wheezing, etc.).
{ (b) Crepitant Sounds.

2. OF THE *Cough*.

The following are some of the more common varieties of Cough :—

- (1) *Cough of the ordinary type*, such as is met with in—(a) Common Colds; (b) Some conditions of mere nervousness; (c) Irritation of certain peripheral nerves, as those of stomach, ear, etc.; (d) Early Phthisis, when, however, it is apt to be specially hacking and irritating; (e) Later Phthisis, when it may vary much in severity, from moderate, though frequent, to severely paroxysmal. Phthisical cough is often succeeded by vomiting.
- (2) *The frequent, prolonged, paroxysmal, and often wheezing Cough* that is characteristic of severe Bronchitis.
- (3) *The frequent, short “suppressed” Cough* of Pneumonia with associated Pleurisy, and of simple acute Pleurisy. It is “suppressed” because it excites pain in the side.

- (4) *The husky and sometimes stridulous or "croupy" Cough* that is characteristic of Laryngitis.
- (5) *The peculiarly "brassy" or ringing Cough* (like the cry of a gander) that is met with in many cases of Aortic Aneurism or Mediastinal Tumour with pressure on the Trachea.
- (6) *The prolonged paroxysm of fully developed Whooping Cough*, with its rapid and long-continued succession of short, sharp coughs, and its final long-drawn stridulous inspiration. A succession of these paroxysms is very often succeeded by vomiting.
- (7) *The loud Barking Cough* met with in some cases of hysteria.

NOTE.—The *Sputum* should be carefully examined and described. Its more common types are—

- (a) Viscid, mucous, and often pigmented, in Common Catarrh; (b) Bloody, in Hæmoptysis; (c) "Nummular," in advanced Phthisis; (d) Copious, frothy, mucous or muco-purulent, in Bronchitis; (e) Sticky, gelatinous, and rusty, in Pneumonia; and (f) extremely fetid in Gangrene of the Lung and some cases of Bronchiectasis.

In special cases a report should be made of the Microscopic characters of the Sputum.

When, with cough, there is no expectoration, *its absence should be noted.*

DISEASES OF THE LARYNX.

Inflammation of the larynx may be acute or chronic.

ACUTE CATARRHAL LARYNGITIS.

Causes.—Contact with irritating vapours, drinking scalding water, impaction of foreign bodies, exposure to damp and cold air, extension of inflammation from other parts, and acute specific fevers.

We have already referred to diphtheria as a cause, and we also pointed out that the only cases, not really diphtheritic, that might be called "croup," were due to acute laryngitis or to laryngismus stridulus in young children.

Pathology.—It is similar to that of bronchitis.

1. Hyperæmia, causing dryness.
2. Exudation of lymph = increased muco-purulent secretion.
3. Changes in the tissues beneath, etc., especially—
 - (1) Œdema of the *submucous coat and the glottis*.
 - (2) Changes in the neuro-muscular apparatus of the larynx.

Symptoms.—Soreness and dryness accompanied by hoarseness, or complete loss of voice. Respiration may be stridulous and noisy. Rhonchi and râles are often abundant.

The loss of the voice is due to failure of the vocal cords to meet in the middle line, and to thyro-arytenoid paralysis.

When the glottis becomes œdematous the symptoms are most alarming. Intense dyspnœa, *dysphagia*, and stridor are the marked features. In children the cough becomes muffled or “croupy.” Death may occur in a few hours. Laryngoscopically: the epiglottis is seen to be much swollen and bright red in colour; the vocal cords may or may not be inflamed.

Treatment.—

Catarrhal Form.—Rest in bed, steam, antiseptic inhalations of eucalyptol or benzoin. A full dose of Dover’s powder often cuts the attack short. Demulcent drinks and absolute rest to the voice.

Œdematous Variety.—Tracheotomy, if the symptoms do not subside in a few hours. This is very seldom necessary in adults.

It should particularly be remembered that very little irritation may provoke œdema of the glottis in gouty individuals.

HOW TO USE THE LARYNGOSCOPE.

1. *Position of Patient*: sitting, body and head erect, knees together, head slightly thrown back.

2. *Lamp*: in line with patient’s ear, nine inches to the left of his head.

3. *Position of Physician*: opposite patient with mirror properly adjusted to head and eye.

4. *Mouth*: wide open.

5. Reflect light upon fauces at correct focal distance of reflector.

6. Warm laryngeal mirror over lamp. Test it against cheek or hand.

7. Direct patient to protrude his tongue.

8. Hold it between thumb and index-finger, in napkin (thumb uppermost).

9. Hold laryngeal mirror like a pen.
10. Place its back gently against uvula.
11. Move your hand slightly towards patient's left, so as to keep it out of line of view.
12. Patient to draw a deep breath, and say "ah," "ur," "eh," or "ee." Be always quiet and gentle; encourage the patient; let each examination be short, even if unsuccessful. Be careful not to hurt patient's tongue, or to burn his mouth, or to push either his uvula or the mirror against the back of the pharynx. Use cocaine to lessen sensitiveness of fauces (KEETLEY).

CHRONIC LARYNGITIS.

Chronic laryngitis may be the sequel of acute laryngitis, but it is much more commonly due to—

1. *Excessive use of the voice*—i.e., "dysphonia clericorum," a condition characterised by an œdematous unhealthy condition of the mucous follicles, which present a raw or even ulcerated condition. Aphonia is very common after prolonged speaking.
2. Tumours, fibroid polypi, epitheliomata, etc.
3. Gout.
4. Various nervous affections—functional and organic.
5. Alcoholism.

Symptoms.—The severity of the symptoms depends on the cause. The more common are—

1. Constant hawking and desire to swallow.
2. Expectoration of muco-purulent phlegm.
3. Attacks of hoarseness or aphonia.
4. Spasmodic breathing (most often associated with irritation by tumours).

Treatment.—Depends on cause. Nervine tonics, such as strychnine and iron, are nearly always indicated along with plenty of fresh air; rest to the voice, and *douching the throat with cold water*. Locally, we may use astringents, such as tannic acid, nitrate of silver (10 grains to ℥j), chloride of ammonium vapour, and various antiseptic sprays.

LARYNGEAL TUBERCULOSIS

Is nearly always secondary to pulmonary tuberculosis, and is due to inoculation by the sputum. It leads to infiltration and ulceration first of the posterior part of the cords and the inter-arytenoid fold, and spreads to the epiglottis and the ventricular bands.

The *symptoms* are those of chronic laryngitis, associated later with dyspnœa, stridor, or pain on swallowing. Coincident enlargement of the bronchial glands may cause paralysis of the recurrent laryngeal nerve.

Treatment.—Insufflations of morphia, curettage or excision of localised swellings, and application of lactic acid (at first 50 per cent, increasing to full strength).

LARYNGEAL SYPHILIS

Is a tertiary lesion. It attacks the epiglottis first, and may spread to the cords and inter-arytenoid fold. It leads to deep ulceration and necrosis of cartilages, and is followed by cicatricial contraction, causing laryngeal deformity.

The *symptoms* are those of chronic laryngitis. Hoarseness is prominent, but there is little cough. Stridor may occur where there is laryngeal stenosis.

The *treatment* is that of tertiary syphilis. Tracheotomy may be required for stenosis.

TUBERCULAR ULCERATION.

1. Attacks posterior wall.
2. Advances slowly.
3. Marked thickening and infiltration.
4. Pain is commonly present.
5. General condition poor.
6. Evidences of phthisis.

SYPHILITIC ULCERATION.

1. Attacks epiglottis.
2. Ulceration is more acute.
3. Little thickening.
4. Little pain.
5. General condition less affected.
6. Evidences of syphilis elsewhere.

LARYNGISMUS STRIDULUS.

FALSE CROUP.

False croup or child-crowing is a spasmodic disease of the larynx, occurring in infants and young children, consisting of a temporary closure of the rima glottidis, causing great dyspnœa and other symptoms dependent on temporary suffocation. There is no catarrhal affection of the larynx.

Ætiology.—Predisposing causes are malnutrition brought about by imperfect feeding ; syphilis, rickets, enlarged tonsils, and the tubercular diathesis. The exciting

Causes are reflex nervous disturbances, brought about by —

- | | |
|---|-------------------|
| 1. Intestinal irritation. | } Sensory nerves. |
| 2. Worms. | |
| 3. Teething (<i>via</i> trifacial nerve). | |
| 4. Exposure to cold (vasomotor). | |
| 5. Overloading the stomach (pneumogastric). | |
| 6. Frights and “starts” (cortical). | |

It will thus be seen that the disease is essentially associated with a neurotic temperament, and disturbances of the various nerve centres. There are three or four varieties of laryngismus. Goodhart describes three forms—

1. *Direct Spasm*, a crowing of a *convulsive nature*, often rachitic.
2. *Infantile Spasm*.—Crowing is due to a congenital valvular formation of the upper orifice of the larynx.
3. *Reflex Spasm*.

Symptoms.—Sometimes a slight cold *precedes the attack* (catarrhal form), but often the child is apparently well on going to bed, and wakes up suddenly at night with a brassy croupy cough and dyspnœa. The spasm is usually worse on first awaking. After a time the spasm passes off and the child, beyond having a slight croupy cough for a day or two, seems

none the worse for the attack. Often a series of paroxysms, however, takes place, or recurs at *night only*. The mother is usually much alarmed, and is very anxious for the arrival of the doctor. The prognosis is favourable, but a fatal ending occasionally takes place.

Pathology.—It is to be regarded as a neurosis at present, but the palate and mucous membrane around the laryngeal orifice are often œdematous.

Diagnosis from Diphtheria, viz.—

1. Absence of false membrane.
2. Absence of marked local inflammation.
3. History.

Treatment.—When in doubt treat as if it were the more severe disease.

1. Steam kettle and medicated vapour.
2. Give a warm bath and apply hot sponges to the larynx. Douche the chest with cold water. Chloroform or amyl nitrite may be inhaled.
3. Bromides in the interval. Any constitutional disease must also be treated.

LARYNGEAL PARALYSIS.

During ordinary respiration the glottis remains partially open, being widened with every inspiration. For the production of voice, the free borders of the vocal cords must be brought almost close to each other in the middle line, only a very narrow chink being between their parallel sides. At the same time the cords must be rendered tense. The narrowing of the chink is brought about by the adductors, viz., the lateral crico-arytenoid, assisted by the arytenoid and *external* thyro-arytenoid muscles. The tightening is due to contraction of the crico-thyroid. The nerves involved are the superior laryngeal and recurrent laryngeal branches of the vagus. It will be easily understood that aphonia or loss of voice may be

brought about by local muscular causes or central nervous lesions. Again the paralysis may be partial, or complete, of a functional nature, or due to organic lesions.

Dr. Gowers gives the following table :—

LESION.	SYMPTOMS.	POSITION OF CORDS.
<i>Total bilateral paralysis</i>	No voice ; no cough ; stridor only on deep inspiration.	Both cords slightly abducted and motionless.
<i>Total unilateral paralysis</i>	Absence of stridor except on deep breathing ; no cough ; voice low and hoarse.	One cord motionless ; the other moving freely, and <i>even beyond middle line in phonation.</i>
<i>Total abductor paralysis</i>	The voice is little changed ; cough normal ; inspiration difficult and long, <i>with loud stridor.</i>	Both cords lie together, and not separated during inspiration.
<i>Unilateral abductor paralysis</i>	Little affection of either <i>voice or cough.</i>	One cord not moving during inspiration.
<i>Adductor paralysis</i>	No voice ; perfect cough ; no stridor or dyspnœa.	Cords are not brought together, but move during respiration.

Causes of Laryngeal Paralysis.

1. From the nerve side.

- (1) Central lesions.—Bulbar paralysis, disseminated sclerosis, etc. •
- (2) Peripheral.—Aortic aneurisms, mediastinal tumours, enlarged thyroid gland, diphtheritic paralysis, all through affecting the recurrent laryngeal nerve.

2. Local lesions.—*Ulceration*, due to syphilis, tubercle, or malignant laryngeal tumours affecting the cords.

3. Functional or hysterical paralysis (nearly always affects the *adductors*).

To sum up. If there be an inability to cough or speak, suspect a serious paralysis. If the *voice is preserved* and the *cough lost*—means unilateral paralysis. If there is normal *cough*, but *no voice*—unimportant. Loud inspiratory stridor means—double abductor paralysis.

Treatment.—Hysterical aphonia must be treated with galvanism. The other forms must be treated on general principles. For detailed treatment consult a good treatise or manual on the Throat.

DISEASES OF THE BRONCHI.

BRONCHITIS.

INFLAMMATION OF THE BRONCHI.

Ætiology.—Occurs most frequently in winter amongst elderly people, but the capillary variety is more often seen amongst young children, especially in connection with whooping-cough and measles; insufficient food, scanty clothing, or on the other hand, excessive confinement in warm rooms, and too warm wrapping up, are the great predisposing causes.

Exciting causes—

1. Spreading of nasal catarrh.
2. Foreign bodies in larynx, etc.
3. Certain infectious diseases, *e.g.*, influenza, measles, diphtheria, etc.
4. Extension of inflammation from other parts.
5. Cardiac valvular lesions.

Pathology.—That of a typical inflammation of a mucous membrane, *i.e.*—

1. Hyperæmia.—The membrane is injected, dry, and secretion scanty.

2. Exudation of lymph with increase of mucus—secretion more profuse.

3. Purulent degeneration of the exudation—muco-purulent phlegm. There is much swelling of the mucosa, leading in the smaller tubes to marked narrowing of their calibre.

4. Changes in the tissues beneath, slight at first, but repeated attacks cause grave changes, *viz.* :—

- (1) Epithelium becomes to a large extent destroyed.
- (2) Muscular coats are fibrosed and rigid.
- (3) Tubes themselves become dilated or varicose, causing (as we shall see farther on) bronchiectasis.

(a) *Acute Bronchitis*.—Ushered in with coryza and pains about the chest, and cough. The pain is burning in character, and situated behind the sternum. The expectoration at first is frothy and scanty, but soon becomes abundant and purulent, and often is expectorated in greenish jelly-like masses; later, it is more muco-purulent. The breathing is much embarrassed, and noisy or whistling in character; the temperature is not high as a rule, and the skin is moist. The urine is of a febrile type, scanty and high coloured. After a few days the more acute symptoms subside, and convalescence becomes rather slowly established. Death is rare in uncomplicated cases, but the disease is often fatal through extension to the bronchioles or air-cells.

(b) *Capillary Bronchitis*.—This variety more frequently affects children, and is characterised by great dyspnœa, *sucking in* of the intercostal spaces, abundant piping rhonchi and small crepitations, high temperature, quick feeble pulse, a tendency to drowsiness or coma, and collapse of the lung. There may be a fibrinous exudation into the bronchioles, which leads to the expectoration of tree-like casts, rolled up in the spit into little glassy balls, which can be readily teased out.

Physical Examination.—Rhonchi and moist râles are heard all over the chest. These vary in character with the size of the tube in which they are produced. If in the large tubes, the rhonchi are deeply pitched and sonorous, and often accompanied by bronchial fremitus. If in the smaller tubes, they are sibilant or piping, the note being higher the smaller the tube. They are heard throughout both respiratory periods. There is no alteration of the percussion note in uncomplicated bronchitis, except where there are collapsed portions of lung; then there are, of course, impaired resonance and absence of breath sounds.

Treatment.—In the first stage employ the bronchitic kettle, containing a solution of eucalyptus or pinol ; administer a brisk saline purge, and a diaphoretic mixture such as—

℞ Pot. Citratis	℥iij.
Vin. Antim. Tart. . . .	℥iij.
Spt. Æther Nit. . . .	℥iij.
Spt. Chloroformi	℥ij.
Aq. ad	℥viij.

Fiat mist. A tablespoonful every three hours for an adult.

Later—ammonia, with senega and ipecac. ; or ammonia and pot. iodid., with the compound tincture of camphor. The strength must be supported by tonics, hypophosphites, etc. Later the mineral acids are of use in diminishing the amount of expectoration.

In the capillary form lowering measures are seldom called for. The two great dangers are—

1. Collapse of the lung.
2. Early heart failure.

If the former threatens, as evidenced by increasing *lividity* and *dyspnœa*, a brisk emetic should be given, followed at once by a stimulant, such as the compound spirit of æther with ammon. carb. Emetics must only be used for vigorous children. In most cases stimulation is necessary, by alcohol or other diffusible stimulant, or by strychnine hypodermically. *Sedatives are nearly always contra-indicated*, as they diminish the expulsive power, causing accumulation of secretion, and hence a tendency to asphyxia.

CHRONIC BRONCHITIS.

(WINTER COUGH.)

Chronic bronchitis may be the result of the acute form ; but it more often results from a series of sub-acute attacks in old people, whereby the bronchial tubes are “devitalised” and prone to inflame on slight provocation, especially in winter time.

Symptoms are dyspnœa, harsh paroxysmal cough, and copious expectoration of phlegm, which varies much in

character, from being mucoid and ærated, to non-ærated muco-purulent, or even pus alone. Chronic bronchitis is too frequently looked upon as a *mere bronchial affection*, forgetting the widespread and grave changes induced, as the following table shows:—

PATHOLOGICAL CHANGES.	PHYSICAL EXAMINATION.	CLINICAL SYMPTOMS.
<p>1. In the tubes themselves — rigidity of walls and subsequent dilatation = bronchiectasis.</p> <p>2. Dilatation of the air vesicles and absorption of their walls, with loss of elastic tissues and capillary vessels = emphysema.</p> <p>3. Hypertrophy of right side of the heart, quickly masked by great <i>dilatation</i>.</p>	<p>1. Abundance of râles. Indistinct signs of cavity.</p> <p>2. Altered shape of chest (barrel-shape), use of auxiliary muscles of respiration, hyper-resonance, decreased breath-sounds and prolonged expiration.</p> <p>3. Increase of cardiac dulness. Epigastric pulsation. Later, signs of tricuspid leakage—<i>i.e.</i>, general venous congestion, venous pulse, and systolic murmur in tricuspid area.</p>	<p>1. Copious expectoration at intervals of fœtid, pus-like secretion.</p> <p>2. Dyspncea through imperfect aeration, increased on exertion.</p> <p>3. Feeble pulse — œdema — perhaps anasarca; in fact all the troubles consequent on general venous congestion of the viscera.</p>

Treatment.—Hygienic measures rank first in the treatment; proper clothing, careful dieting, and change of air do more good than drugs. Of drugs, the iodides with ammonia are useful, terebene is praised; digitalis and strychnine are extremely beneficial when the pulmonary circulation is more sluggish than usual. Diuretics may be used to relieve passive congestion. If this is present, rest in bed is imperative.

BRONCHIECTASIS.

Dilatation of the bronchi has already been referred to, as a complication or result of bronchial disease. We must now consider more fully how the condition is produced.

1. It may be a congenital defect, or produced by—
2. Primary disease of the bronchial walls.
3. Contraction of lung tissue in phthisis, etc.

Two forms occur :—

1. Cylindrical or fusiform.
2. Saccular.

The former is more often produced by strain from within, such as violent coughing, the rigid walls yielding to the expiratory intra-pulmonary pressure.

The saccular form is most often produced by—

1. Contraction of new fibrous tissue outside the tubes.
2. Breaking down of the lung substance, causing diminished support, *and yielding of the walls at that point.*

The dilated portion is usually smooth, until ulceration from retained secretion occurs. The walls are very thin, the muscular and elastic tissue being much atrophied.

Symptoms.—Physical examination may reveal all the signs of a cavity. The expectoration is copious and foetid, and if allowed to stand it separates into three layers, the lower layer being almost pure pus; next, a more granular zone; and a clear frothy layer on top of this.

Microscopically it shows—

1. Pus cells and epithelial debris.
2. Crystals of fatty acids.
3. Elastic fibres (may be absent).

The horrible odour is due to valerianic and butyric acids, H_2S , etc. The mode of expectoration is characteristic, the patient usually bringing up a huge quantity in the morning, or on moving after resting in an horizontal position for some time.

Marked emaciation, accompanied by irregular fever and night sweats, is usually present. Clubbing of the fingers and toes is marked. Slight hæmoptysis may be present, and fatal hæmorrhage has occurred from rupture of an aneurism in the walls of a cavity.

Treatment.—Antiseptic inhalations, capsules of creosote, etc. Terebene is warmly praised by some. Incision and drainage of the cavities has sometimes proved successful.

ASTHMA.

Asthma is a disease characterised by sudden attacks of paroxysmal dyspnoea, which subside after a time, but tend to recur at intervals.

Ætiology.—Heredity is a most important factor. When it develops in children there is usually a history of post-pharyngeal trouble, measles, whooping-cough, or imperfect recovery after capillary bronchitis. There may be a family tendency to gout or phthisis, or to nervous complaints. Probably *most* cases have a neurotic origin. It is much more frequent in males than in females, possibly because of their greater liability to bronchial affections generally. The exciting causes are either direct irritation of the bronchial mucosa, or indirect irritation through the nervous system or the blood.

Pathology.—All authorities agree that the affection is due to diminished calibre of the smaller bronchioles, but the nature of such contraction is still disputed. The most probable theory is the first, viz.:—

1. *Reflex* spasm of the bronchial muscles, associated with hyperæmia and turgescence of the mucous membrane lining the smaller bronchioles, and the exudation of a characteristic mucus.

2. *Hyperæmic swelling of the mucous membrane*, like “nettle rash” (Sir A. CLARK), due to a vasomotor dilatation.

Possibly both theories are necessary to explain all cases.

Symptoms.—

1. *Premonitory Symptoms.*—There is usually some visceral disturbance such as flatulence, etc., but the onset may be quite sudden.

2. *The Attack.*—It occurs most frequently during the night, the patient waking up with a feeling of great dyspnoea; he

feels as if there were not sufficient air in the room, and asks for the windows and doors to be opened. The characteristic attitude of asthma is assumed—*i.e.*, the patient grasps some support to fix the shoulder girdle, in order to bring his extraordinary muscles of respiration into play. Expiration is prolonged. Sibilant rhonchi cause a peculiar, noisy, pipe-like wheezing; but in spite of the extraordinary efforts little air enters the lung. A paroxysm of coughing and expectoration gives slight relief or even terminates the intense dyspnoea, and sleep may supervene, or a slight lull may be succeeded by another paroxysm.

The Sputum is usually expelled with the greatest difficulty, and is distinctly peculiar in its composition. The ball-like gelatinous masses can be unfolded, and are then found to represent casts of the small bronchioles.

Curschmann describes the expectoration under the microscope as follows:

At First.—The pellets show two forms of spiral threads (Curschmann's spirals). The one form entangles within its meshwork cells in various stages of fatty degeneration. The other form contains a central clear filament, surrounded by a network of other filaments.

Later.—The filaments disappear, and octahedral crystals of phosphates appear in the now muco-purulent expectoration (Charcot-Leyden crystals). The course of the attack depends on the immediate cause, and the amount of bronchitis associated with it. As a rule, the asthmatic attacks tend to become less severe, but the bronchial affection more pronounced. Death seldom or never takes place from pure asthma.

Physical Signs.—

During the Attack.—The thorax is expanded and fixed. The diaphragm moves but slightly, inspiration is short, and expiration prolonged. Auscultation: vesicular breathing is drowned by sibilant rhonchi, or later, by bubbling râles. Percussion reveals marked hyper-resonance (acute emphysema). The cardiac and hepatic dulness are diminished. This condition

disappears at the end of the attack, but after many recurrences tends to merge into permanent and chronic emphysema.

In the Intervals.—There may be the usual signs of bronchitis over the lungs, or but little departure from the normal.

Treatment.—

During the Attack.—Remove any obvious cause of reflex irritation, such as an overloaded stomach. A brisk emetic often cuts an attack short; nitrites, especially nitrite of amyl, or chloroform may be inhaled. Belladonna, stramonium, and lobelia in combination with ammonia are useful remedies; the fumes of burnt nitre papers, etc., are also recommended.

The drug which has most claim to be regarded as specific is iodide of potassium. It should be given during the intervals and steadily persevered with. It is often successful, but as often fails, the disease being very refractory to treatment.

During Convalescence.—Change of air, careful diet, cod-liver oil, tonics, etc.

DISEASES OF THE LUNGS.

EMPHYSEMA.

By emphysema of the lungs is meant—(1) A condition where the air-cells are over-distended = vesicular emphysema; (2) A condition where the air has accumulated in the interstitial tissue = interstitial emphysema. Only the former is here described.

Emphysema is again divided into—

1. *Compensatory Emphysema.*—A condition where a portion of lung expands to take the place of a collapsed portion; seen in catarrhal pneumonia, pleuritic adhesions, in areas of old cicatrices, etc.

2. *Atrophic Emphysema*, or small lung emphysema, due to senile atrophy. The chest is of course small.

3. *Hypertrophic or ordinary Emphysema.*—A condition characterised by—

(1) Over-distension of the vesicles.

(2) Atrophy of their walls.

- (3) Obliteration of blood-vessels, and a consequent diminished "oxygenating" area.
- (4) Changes in the shape of the chest.
- (5) Changes in the heart (right).
- (6) Changes generally, due to imperfect exchange of gases between the blood and the air.

Causes.—They include all those factors which keep up a more or less persistent high intra-alveolar tension.

1. Playing on wind instruments.
2. Certain occupations, such as glass-blowing, colliers, etc.
3. Chronic cough.
4. Dr. Jackson of Boston lays great stress upon the hereditary character of emphysema. This is probably due to congenital weakness of the elastic tissue of the lungs.

Pathology.—The pleuræ are pale. The lungs pit readily on pressure, and have a peculiar soft downy feel. The dilated vesicles are well seen on the surfaces, and also projecting from *the free margins* of the lungs. The vesicles first become dilated, then coalesce by atrophy and absorption of the septa between the neighbouring cells. The capillaries in the affected area disappear. Though the changes are principally in the lung vesicles, there are always more or less marked changes in the bronchial tubes. Bronchiectasis is often a marked feature.

Symptoms.—They are too well known to require details; the important points to remember are—deficient aeration means *Dyspnœa* and cyanosis; the retention of waste products within the blood means impure blood and defective nutrition; the loss of capillary vessels is followed by increased resistance in the pulmonary circulation, leading to hypertrophy and afterwards to dilatation of the right heart. This causes general venous congestion, dropsy, and cyanosis. Patients are often ill-nourished and emaciated.

Physical Signs.—Barrel-shaped chest, prominent sternum, deep sternal fossa, prolonged expiration, and hyper-resonance are the more common features. The auxiliary muscles in the

neck, etc., are all employed in respiration to increase the air capacity of the chest. The areas of cardiac and hepatic dulness are encroached upon, and the margin of the lung becomes fixed in the position of full inspiration, from the disappearance of the elastic tissue. A zone of dilated venules along the attachment of the diaphragm is spoken of by some writers. The epigastric pulsation and altered position of the apex beat are important signs.

Treatment.—That of chronic bronchitis. Sudden and grave attacks of dyspnoea may be treated by inhalation of nitrite of amyl, venesection, etc. Where the heart is beginning to fail, strychnine, and later digitalis, may be required.

COLLAPSE OF THE LUNG.

Collapse of the lung may be of four types—

1. *Atelectasis*.—A condition found in weakly new-born children, when there is not sufficient inspiratory power to inflate the alveoli. The collapsed patches are of a slate colour and sink in water.

2. The form due to pressure from pleuritic effusion. The *whole* lung may collapse.

3. Collapse due to wounds of the chest-wall, and perforation of the pleura. In this form the lung at first is congested, but finally becomes anæmic.

4. Ordinary or lobular collapse, as seen in broncho-pneumonia. Two theories are advanced to explain this condition—

- (1) That a bronchus is plugged by a pellet of mucus which acts as a ball valve—*i.e.*, allowing air to pass *out*, but none to *enter*.
- (2) That the plug does *not move at all*, but merely prevents access of *air* during inspiration. The alveolar air is exhausted by absorption, and none being taken in to replace it, the lobule collapses.

Collapse may occur where there is no plug, if there be paralysis of the respiratory movements. This is due to the

elastic recoil of the lung tissue, aided by absorption of air by the blood-vessels.

Symptoms.—If the collapsed area be extensive, any already existing dyspnœa is increased, the pulse becomes rapid, and there may be cyanosis; in slight cases the symptoms are merely those of the primary disease. The physical signs are subcrepitant râles, weakened breath-sounds, and if the area be extensive, dulness and possibly tubular breathing. Collapse occurs often in measles, whooping-cough, and other conditions which give rise to catarrhal pneumonia.

Treatment.—Depends on the cause. Emetics and diffusible stimulants.

ŒDEMA OF THE LUNG.

Definition.—An accumulation of serous fluid in the interstices of the lungs, air vesicles, and bronchioles.

Causes.—Nearly always associated with general blood diseases, such as Bright's, the anæmic diseases, etc. It is common in the later stages of—

- | | |
|---|--------------------------|
| 1. Valvular disease of the heart. | |
| 2. Malignant fevers. | |
| 3. Paralysis. | } Hypostatic congestion. |
| 4. Long-continued rest on the back. | |
| 5. <i>After the use of pilocarpine.</i> | |

Pathology.—The lung is heavy and bulky. On pressure it pits, and a quantity of blood-stained serum exudes.

Physical Signs.—The breath-sounds are deficient, and masked by fine râles. The percussion note may be resonant at first, but afterwards becomes dull at the bases. The explanation given is that a lung, when *partially collapsed* outside the body, gives forth a resonant sound, *due to diminished tension of the lung tissue.*

Symptoms.—Those of serious pulmonary embarrassment (already described under collapse of the lung), *plus* the abundant expectoration of the frothy serum.

Treatment is that of the cause.

PNEUMONIA.

By pneumonia proper is meant an acute specific fever associated with consolidation of the lung. Three types are usually described, viz.:—(1) Croupous, lobar, or acute pneumonia (pneumonia proper); (2) Catarrhal or lobular pneumonia (broncho-pneumonia); chronic interstitial pneumonia (cirrhosis or fibrosis of lung). The two latter are diseases quite distinct from true pneumonia.

CROUPOUS OR ACUTE PNEUMONIA.

This form is characterised—pathologically: by an inflammation giving rise to an exudation, *rich in fibrin, and showing colonies of special organisms—i.e., pneumococci*;—clinically: by its abrupt onset, by running a definite course, and ending by crisis. It usually begins at the base, and may involve one or more lobes.

Ætiology.—Croupous pneumonia is a most common affection, occurs most frequently in winter and spring, and attacks all ages, though it is more frequent before the age of ten, and between twenty and thirty, than at other ages. Amongst the more common exciting causes are draughts, intemperance, exposure to inclement weather, or irritating gases. Frequently this kind of pneumonia occurs in epidemic form; indeed, this form is now looked upon as a “*specific fever with marked lung symptoms*.” It is, however, described here, instead of among the specific fevers, as the characteristic signs and symptoms are mainly pulmonary.

Pathology.—It is convenient to describe four stages, viz.:—

1. Hyperæmia or engorgement.
2. Red hepatisation.
3. Grey hepatisation.
4. Resolution.

1st Stage.—The lung is injected, heavy, and more friable; on pressure, there exudes a frothy serum tinged with blood and slightly aerated. The lung still floats in water.

2nd Stage or Red Hepatisation.—The part involved is solid, presents a granular or red granite appearance, and sinks in water. The alveoli are filled with a coagulated exudation, which shows under the microscope—

1. Fibrin.
2. Proliferated cells.
3. Leucocytes.
4. Red corpuscles.
5. Granules and cell debris.
6. *Pneumococci*.

3rd Stage or Grey Hepatisation.—The lobe has now the appearance of grey granite, the lung substance is softer and more friable; on pressure, a dirty purulent fluid exudes. The grey appearance is due to four factors—

1. Decolorisation of the red blood corpuscles.
2. Obliteration of the alveolar blood-vessels from pressure.
3. Fatty degeneration of the coagulated material.
4. Great infiltration of leucocytes.

A more advanced stage, in which the lung tissue is bathed in purulent fluid, is known as purulent infiltration. It is probably inconsistent with life.

4th Stage or Resolution.—Means resolution of the inflammatory exudation, principally by absorption, but partly by liquefaction and expectoration.

Pneumonia may affect a lobe, or the whole of a lung, or it may attack both lungs. Double pneumonia occurs in about ten per cent of cases. Different parts of the same lung may at the same time show different stages. There is always some degree of pleural inflammation over the affected area.

The Germ or diplococcus pneumoniae of Fränkel consists (in cultures) of *cells arranged in pairs, and sometimes in chains*. In the tissues the microbes become lancet-shaped. They retain the aniline stain when treated by Gram's method, Friedländer's pneumobacillus, streptococci or staphylococci, may cause secondary pneumonia, usually lobular.

Physical Examination.

1st Stage.—*Percussion* yields a slight dulness, but sometimes even a slight hyper-resonance may be present. *Auscultation* reveals the characteristic fine crepitations, compared to the sound produced by the rubbing together of hair between the fingers. This fine crepitus is due to the separation of the moist surfaces of the alveoli from each other. It is therefore heard towards the end of inspiration, and not during expiration.

2nd Stage.—Gives the signs of consolidation, viz.—

Inspection shows diminished movement.

Palpation confirms this, and also demonstrates *increased* vocal fremitus.

Auscultation reveals the absence of vesicular breathing, but presence of typical tubular breathing. *Vocal resonance* is increased to the pitch of bronchophony, or even whispering pectoriloquy.

Percussion yields a dull note (not absolutely so flat as in pleuritic effusion).

3rd Stage.—This stage of grey hepatisation can scarcely be differentiated by physical examination from that of red hepatisation.

4th Stage of *Resolution*.—Here we have a speedy clearing up of the exudation, and a return to the normal condition. Small râles or *coarse* crepitations (*crepitus redux*) are heard, the dulness is less marked, and the movement increased.

Symptoms.—Croupous pneumonia may be ushered in with one or more rigors, *rapid* rise of temperature, and localised pain, plus the ordinary accompaniments of the febrile state. As the disease progresses, several characteristic features develop. The pain becomes less marked, but there is greater dyspnoea, and a marked disproportion *between respiration and pulse*. The former may be from thirty to seventy per minute and the pulse perhaps only 110. The cough becomes either hacking or paroxysmal in character, and there is expectoration of the *rusty, viscid phlegm, pathognomonic of this condition*. The pneumonic countenance develops—*i.e.*, flushed face, malar lividity, dilated

pupils, and crops of herpes round the mouth. The urine is highly febrile, *chlorides are markedly diminished* and urates increased. Between the fifth and eighth day the symptoms most frequently *abate quite suddenly*, and rapid recovery takes place; but often, instead of this happy termination, the temperature increases, or perhaps falls to sub-normal, the *pulse becomes more rapid*, tongue dry and brown, the sputum less viscid and prune coloured, the patient quickly falls into the typhoid state, and death takes place most frequently from heart failure. Sometimes prolonged exhaustion, or œdema of the sound lung brings about a fatal issue. A *pseudo-crisis* is common about the fourth or fifth day, the temperature falling rapidly to normal, but *the pulse does not fall with it*. In twenty-four hours the temperature may be as high as ever. At the true crisis, pulse and temperature fall together, usually about the eighth to tenth day, but some cases may abort earlier.

Special Points to Note—

1. The *viscid rusty phlegm* (if it becomes prune-coloured, is an extremely bad sign).
2. Marked disproportion between pulse and respiration.
3. Diminished chlorides in the urine.

As failure of the right side of the heart is a common cause of death, special attention should be paid to the state of the pulse, and condition of the second sound of the heart in the pulmonary area.

Treatment depends entirely on the type of case, and condition of the patient. In no disease has there been more serious blundering; and routine treatment is the worst of all treatments. Answer the following questions before prescribing. Is the patient full blooded, and is there a full bounding pulse? Is the pulse feeble, irregular, or intermittent?

In the first case, in a young and previously healthy adult, if there be cyanosis, or signs of dilatation of the right heart, blood-letting to the extent of a few ounces may perhaps relieve the strain upon the right heart, but more generally treatment

should be directed to maintaining the strength from the outset.

In the latter case we can hope for nothing from a depressing treatment, so stimulants must be resorted to, such as alcohol, ammon. carb., egg and brandy mixture, quinine, æther, etc. When the sputum is very bloody, I have seen nothing act as well as twenty minims of tinct. ferri perchlor., with five grains of quinine every four hours or even oftener. When there is evidence of failure of the heart (weakness of 2nd pulmonary sound, etc.) digitalis should be resorted to. Many prescribe it from the outset. *Chloral* should be avoided.

The diet should consist of milk, beef-tea or broths, white of egg, and so on. The patient should be as little moved as possible, and the bed-pan must be used.

Remember also that narcotics are not well borne in respiratory embarrassment as a rule, but *if pain be excessive* a hypodermic injection of morphia does *more good* than harm, notwithstanding that theoretically morphia is contra-indicated. It should not be given later than the first few days of the illness. Poultices may be applied to the chest, but they are of doubtful use where they are carelessly made. Some authorities advocate the local application of ice. Cold packs applied to the trunk only, and frequently repeated, are very useful in relieving both pain and fever. Depressant antipyretics are to be avoided.

BRONCHO-PNEUMONIA.

(CATARRHAL PNEUMONIA.)

Catarrhal Pneumonia, as its name implies, is usually due to extension of inflammation of the bronchioles into the air vesicles. It may be caused by infection through the blood. *Frequently the process is set up in collapsed lobules.*

Ætiology.—It is most frequently seen amongst children, old people, and those who have to assume the recumbent position. Perhaps this, more than any other affection, brings about a fatal issue in the specific fevers of childhood. It is a very frequent accompaniment of whooping-cough and measles.

Causes.—The more common are—

1. Chills and exposure to inclement weather.
2. Extension of bronchial affections.
3. Inhalation of irritating vapours, gases, etc.
4. As a part of the tuberculous process.
5. As a sequence of infectious fevers.

Pathology.—We find consolidated patches, *lobular*, indefinite, or sometimes sharply defined. The *air vesicles* in the consolidated area have congested walls and swollen epithelial lining. They are filled with

1. Proliferated epithelial cells in abundance.
2. Leucocytes.
3. Mucus, but as a rule no fibrin.

The bronchioles are inflamed, frequently plugged, and their walls infiltrated with small cells.

The surrounding lobules are somewhat congested, and emphysematous (compensatory emphysema). Collapsed areas may also be found.

Symptoms.—At first merely those of bronchial catarrh; the temperature rapidly becomes high, and markedly oscillating, or irregularly remittent in character. The dyspnoea becomes marked; the pulse rapid, feeble, and irregular; the cough harsh, short, and painful; the sputum is scanty, *never rusty*, but may be streaked with blood. The disease ends by lysis, and frequently the exacerbations of fever after the temperature has become normal, are most numerous. Loss of flesh is marked, and difficulty might be experienced in diagnosing this affection from acute tuberculosis. Catarrhal pneumonia is sometimes followed by phthisis. Brunton's explanation is: "That the delayed resolution of the inflammatory products forms a suitable nidus for the tubercle bacillus," and he further suggests the use of arsenic to hasten fatty degeneration and absorption. Death often takes place from asthenia; or recovery is followed by a rather tardy convalescence.

Physical Signs are very uncertain. The chief signs over the consolidated areas are, increased vocal resonance and

fremitus, *slight* tubular breathing *with small râles*. There may be scattered patches of dulness, usually only relative, but never anything like lobar condensation. The intercostal spaces are often sucked in over the collapsed areas.

Treatment.—*Avoid lowering treatment.* Give ammonia, senega, and ipecac. Quinine, whisky, egg mixture, are all useful; also the use of antiseptic inhalations. *Great care should be taken during convalescence.* Woollen clothing, cod-liver oil, hypophosphites, and malt extract, etc., must not be forgotten.

Interstitial pneumonia will be described in connection with the subject of phthisis.

PLEURISY AND EMPYEMA.

Pleurisy is an acute inflammation of the lining membrane of the lungs and thorax.

Causes.—

1. The so-called primary or idiopathic pleurisy has been shown in very many instances to be of tuberculous origin. Certain cases arise apparently from chill, but even in these tuberculous affections of the lung are often found.

2. Secondary pleurisy may arise from numerous causes:—

- (a) Pneumonia.
- (b) Various specific fevers.
- (c) The final stages of chronic diseases (cirrhosis of the liver, Bright's disease, cancer, etc.).
- (d) Irritation from tumours—*i.e.*, aneurisms of aorta, tumours of lung, mediastinal glands, etc.
- (e) Traumatisms, and rupture of abscesses into the pleura.

Pathology.—That of a typical inflammation of a serous membrane, viz.—

- 1. *Hyperæmia*—loss of lustre, membrane is dry and red.
- 2. *Exudation of lymph*, which coagulates, and gives a shaggy appearance to the membranes.

3. *Effusion of fluid* which is sero-fibrinous, yellowish-green in colour, with floating flakes of lymph. Specific gravity 1010 to 1020; coagulates on boiling (from amount of albumin); it also contains a large amount of fibrin. It may be hæmorrhagic in tuberculous or malignant pleurisy.

4. *Resolution*, with more or less permanent *adhesions*.

The process, however, may stop at the second stage, and the exudation speedily resolve—*i.e.*, *dry pleurisy*; or the effusion may become purulent, and constitute an empyema.

Effects of the Effusion.—If large, it produces grave symptoms by the pressure on the lung tissue and viscera near.

1. It causes collapse of a portion, or sometimes the whole of the lung. (The lung naturally *tends* to collapse, by virtue of its own elasticity.)

2. The lung may be actually *pushed* across the middle line.

3. In extreme cases the heart, great vessels, and mediastinum are pushed to the *opposite side*.

The thorax bulges *forward*, and the liver or spleen get displaced *downwards*. The intercostal spaces may be slightly distended, and the diaphragm much embarrassed.

Absorption should occur in from 9 to 21 days. If the fluid is unusually slow in absorbing, suspect empyema.

Are there any absolute signs of the fluid being purulent? No; but there are usually *some* signs, such as—

1. Period of absolute dulness persisting.
2. Temperature becoming septic in character.
3. Rigors.
4. Night-sweats. (Patient often complains that his night-clothes are wet through.)

The only certain way of ascertaining the presence of pus, however, is to use a hypodermic needle as an aspirator.

Symptoms.—Pleurisy is ushered in by—

1. Slight sense of chilliness. Rigor is uncommon.
2. Rapid ascent of temperature (which is not so high as in pneumonia).

3. Lancinating, tearing pain in the side, rendered worse by *any respiratory act*. The pain may be referred to the epigastric or umbilical region.

As the disease advances, and effusion takes place, the severe pain becomes replaced by dyspnœa. The patient lies on the affected side to give the sound lung more freedom. The respirations are hurried, pulse quickened, and cough is hacking in character; the sputum is, however, slight in amount, mucoid in character, and *never rusty*, unless pneumonia exists as a complication. The course of pleurisy is longer than that of pneumonia, and the fever terminates, in favourable cases, by lysis, not crisis. The malar flush is absent, and the general symptoms are less severe in cases of moderate effusion. Death may occur from collapse of the lung, or through the advent of some—

Complications such as—

Empyema.
Pneumonia.
Meningitis.
Heart failure.

Physical Signs.—*Vary with the stage.*

1. *Stage of Fibrinous Exudation.*

- (1) Auscultation reveals the characteristic friction rub. This sound, synchronous with the chest movements, is usually leathery-creaking in character, but is sometimes quite musical, like rubbing a pane of glass with a moist finger.
- (2) Palpation yields friction fremitus.

2. *Stage of Effusion.*—The physical signs over the affected area are—

- (1) Absence of breath sounds.
- (2) Absence of vocal resonance and fremitus.
- (3) Marked dulness on percussion, and board-like resistance.
- (4) Diminished movement.

3. *Above the line of Effusion.*

Just above is a small area where the voice is transmitted through—

- (1) Slightly *condensed* lung ;
- (2) A *thin* layer of effusion ; consequently giving rise to a peculiar modification of the voice, termed ægophony—compared by some writers to the bleating of a goat.

4. Over the *lung* above the effusion.

The signs will depend on the amount of pressure.

Usually, we get on—

Inspection—Diminished movement.

Palpation—Vocal fremitus increased. Vocal resonance increased to the extent of bronchophony.

Percussion—"Skodaic resonance." If heavily percussed, we get almost a "cracked-pot" sound.

Sometimes in the effusion stage, over the fluid, the breath sounds are *replaced by slight tubular breathing* (OSLER). This, however, must be due to collapse of lung tissue around *patent* tubes, or a *patch of pneumonia* ; and cannot, therefore, be looked upon as a *sign of effusion*, but of a *complication*. I earnestly ask the student to get a thorough understanding of (1) how breath-sounds are *produced*, and (2) what the necessary conditions are to *modify those sounds—then*, reason for himself what "ought to occur under certain conditions." If we speak of possibilities of what *might* be heard in effusion, it would fill a book, and surely "*typical*" signs are enough for a junior student to get up intelligently.

Signs of displaced organs—

1. Altered position of "apex beat," and displacement of the cardiac dulness to right or left according as the pleurisy is left- or right-sided.
2. Ensiform cartilage pushed aside.
3. Liver pushed downwards.

Empyema—special points—

1. Whilst an empyema is frequently due to the *pleuritic effusion* becoming purulent, it may be *primary*—*i.e.*, purulent from the beginning.

2. Note that when “pleurisy” is a *sequela* or *complication* of the *infective fevers* or *pyæmic state*, it *nearly always becomes purulent*.

3. If pleurisy is a result of pneumonia, cancer, or tubercle it is usually *purulent from the start*. In malignant pleurisy the effusion is generally hæmorrhagic.

4. Dr. Cathcart has called attention to the peculiar state of the ribs in *old empyemas*, viz.—

(1) A crowding together of the lower ribs.

(2) Absorption of a portion of rib (atrophy), with a deposit of new bone (hypertrophy), giving the rib on section a Δ appearance—*i.e.*, atrophy at apex, and hypertrophy at base.

5. The prognosis is much more unfavourable, and demands surgical interference, as *pus may burrow anywhere*.

Treatment of pleurisy.

Onset.—General principles of “fever.” Relieve the intense pain by—

1. Poultices or blisters.
2. Hypodermic injection of morphia, or
3. Fifteen grains of pulv. ipecac. co.
4. Strapping the affected side (to diminish movement).
5. Leeches.

During Effusion.—Salines, quinine, digitalis, and ammonia.

To Promote Absorption.—Pot. iodid., ammonii carb., ammonii chlorid., arsenic, and iron. Externally lin. iodi., flying blisters, or Ung. Hydrarg.

During Convalescence.—If slow, nourishing diet, cod-liver oil, arsenic and quinine, or phosphorus.

If the fluid is not rapidly absorbed, even when it is shown to be non-purulent, it should be removed by aspiration, which

may be repeated on reaccumulation. Aspirate at once if there are signs of serious pressure on the heart or interference with breathing, and always if the dulness reaches the level of the second rib in front.

Pleurisy occurring in the young adult is often associated with, or develops into phthisis.

Empyema.—

1. Evacuate pus—by—

(1) Aspiration.

(2) *Resection and free drainage.*

2. Tonics ; remember the tendency of this complication to be associated with tuberculosis.

Diagnostic Table from Dr. F. Roberts's Hand-book.

(MODIFIED.)

	BRONCHITIS.	CROUPOUS PNEUMONIA.	CATARRHAL PNEUMONIA.	PLEURISY.	ACUTE PHTHISIS.
1. Mode of invasion.	Coryza, and other symptoms of "cold." No marked rigors, but only slight and repeated chills, if any.	One or more severe rigors. Often vomiting.	Generally after bronchitis, or collapse, and without distinct rigors.	Frequently none, but sometimes several, not severe rigors.	Follows pneumonia, bronchitis, or catarrhal pneumonia; or begins with severe rigors, often repeated.
2. Sensations about the chest.	Soreness, heat, etc., behind the sternum. Muscular pains from cough.	Pain in the side at first, not stitch-like, but more dull and diffused.	Pains about the chest, but not specially localised.	Severe, stitch-like pain in side, increased on respiratory movements.	Generally pains in various parts of the chest.
3. Cough.	In paroxysms, often severe.	Hacking, or in paroxysms.	Short, hacking, and painful.	Slight, and patient tries to repress it.	Frequent and violent fits.
4. Expectoration.	Abundant muco-purulent, etc., changing its characters as the case progresses.	Considerable; viscid, tenacious, "rusty."	Often less than before; not "rusty."	Absent, or very slight, and of no special characters.	Abundant, perhaps purulent, or nummulated; often streaked with blood.
5. Disturbance of breathing.	Sense of dyspnoea, in proportion to the extent of the disease; may be extreme. Pulse-respiration ratio not proportionately altered.	Very rapid breathing, and much perversion of pulse-respiration ratio, but not proportionate feeling of dyspnoea until the later stages, when dyspnoea is marked.	Rapidity of breathing increased when it occurs in bronchitis; dyspnoea may be marked.	Quick, shallow breathing at first, but less disturbance of pulse-respiration ratio than in pneumonia. Later on, more or less actual dyspnoea according to amount of pressure.	Great dyspnoea, and very hurried breathing.

6. Degree of pyrexia.	Often absent or slight, and temperature rarely above 100° to 102°. Skin moist.	Considerable; temperature usually high, but there are considerable remissions, at irregular intervals.	Temperature high, but course of temperature. Skin not acridly hot.	Often very high, but no regularity in temperature, <i>i.e.</i> , oscillates, but usually higher in evening.
7. Aspect of patient, and general condition.	Tendency to cyanosis, if the disease be extensive. In some cases adynamic symptoms.	Marked flushing of face, often unilateral. <i>Malar</i> cyanosis. Herpes round mouth.	Nothing special. No particular prostration, or tendency to cyanosis unless dyspnoea becomes marked.	Severe prostration and weakness, with profuse night-sweats.
8. Physical signs.	Various râles and bronchial fremitus. Signs of obstruction of bronchial tubes. More or less bilateral.	At first, fine crepitations, followed by signs of consolidation, <i>viz.</i> , diminished movement, increased vocal fremitus, dullness, bronchial or tubular breathing, increased vocal resonance, etc. Usually one base is affected. The side is not notably enlarged, nor is there displacement of organs.	At first friction-sound or fremitus, succeeded by signs of fluid, <i>viz.</i> , side often enlarged, movements interfered with, diminished vocal fremitus, dullness occasionally movable, weak or suppressed breathing. Usually on one side, and often displacement of organs.	At first merely signs of bronchitis, followed by consolidation, softening, or excavations in different parts. There is frequently nothing but scattered râles in very acute cases.
9. Course and termination.	Variable. No crisis. Tendency to death by apnoea or adynamia in the capillary variety.	(1) Often a marked crisis from 5th to 8th day; (2) death; (3) gangrene of lung; (4) abscess of lung.	No crisis, and course very variable.	Generally very rapid course, and fatal termination.

PHTHISIS.

(*φθίνωμαι*—I waste.)

By phthisis we mean a morbid condition of the lung tissue due to the invasion of a specific bacillus ; and characterised pathologically by formation of tubercles, which subsequently undergo retrograde changes, involving destruction of lung substance.

Ætiology.—While phthisis is not in the strict sense hereditary, it is certainly true that consumption does affect some families generation after generation. It is probable that whilst the disease is not strictly hereditary, a *predisposition* to it *is*. Or putting it more plainly, whilst the offspring of consumptive parents are not *born with the disease*, they are born with a highly suitable soil for its development. But even in such cases, it is obvious that children not hereditarily predisposed may acquire it by transmission from infected parents. The more common causes tabulated are—

Causes.—

1. Hereditary predisposition.
2. Bad sanitary surroundings.
3. Deficient food.
4. Living on damp soils.
5. Undue exposure.
6. Certain occupations—stone-masons, knife-grinders, etc.
7. Excessive lactation.
8. Result of exhausting diseases, especially catarrhal pneumonia.

We have at page 93 considered the main facts relative to the bacillus, and the peculiar manner in which it exerts its pernicious influence. We have only to apply to the lung that which we have already seen occurs in tissues generally, to get a fair picture of a typical case of pulmonary consumption.

PATHOLOGY.	PHYSICAL SIGNS.	CLINICAL SYMPTOMS.
1st stage. { 1. Damage to the lung tissue and invasion of the bacilli.	Weakened breathing; prolonged expiration, cog-wheel inspiration, etc., <i>plus</i> adventitious signs of the particular exciting cause.	Possibly no symptoms beyond a slight persistent cough, weakness, anorexia, etc.
2nd stage. { 2. Inflammatory changes around bacilli, resulting in the formation of grey tubercles. 3. Inflammatory zone around the <i>tubercles</i> .	{ Consolidation. Diminished movement. Slight flattening. Dull note. Vocal fremitus, and Vocal resonance increased. Bronchial breathing (may be " <i>tubular</i> " in quality).	Increased weakness and cough; elevation of temperature, especially in the evening; diffuse pain in chest.
3rd stage. { 4. Commencement of breaking down. 5. Formation of cavities.	Consolidation, but attended with <i>moist sounds</i> . Flattening is marked; movement much diminished; boxy note or cracked-pot sound; whispering pectoriloquy; amphoric or cavernous breathing, <i>plus</i> adventitious sounds.	Increase in the severity of above symptoms; there are now great emaciation, night-sweats, oscillating temperature, hæmoptysis, distressing bouts of coughing, characteristic sputum, diarrhoea. Evidence of lardaceous disease in other organs, especially <i>liver</i> .

Whilst the above table gives a short summary of the various aspects of this disease, it must in addition be remembered that cases widely differ. For instance, the disease may commence with very marked symptoms of bronchial catarrh; others again are so insidious in their onset that the greater part of a lung may be consolidated before any severe symptoms develop. Severe hæmoptysis may be the first symptom, or the disease may commence as a pleurisy with effusion. And so with the *physical* signs, they vary enormously. We have, it is true, sketched the typical signs of *typical* damage to the elasticity of the lung, of consolidation, and of a cavity; but such typical signs are often absent or masked by the bronchial affection; indeed, the student, who has a clear idea how the various sounds are produced, will *learn more* by carefully examining six cases of phthisis than he will by digesting the most admirably written treatise on the subject. We shall now consider the various symptoms in detail.

Onset.—The disease usually commences at the apex of the lung by the formation of tubercles in the peri-bronchial tissue, but gradually deposits of tubercles form at lower levels, especially along the anterior margins. When the lower lobes are affected, the seat of invasion is about that part of the lung corresponding to the vertebral border of the scapula, when the hand of that side is hooked over the opposite shoulder. Owing to the peculiar manner in which the disease begins and progresses, it is possible in *one* case to get the signs of invasion of tubercle at the *base*, consolidation in the *middle*, and excavations at *the apex*.

Course.—The disease may be rapid or extremely chronic. It may be hastened by severe complications, bad treatment, unusually suitable soil, or putrefaction of cavity contents. It may on the other hand be arrested or hindered by—

1. Formation of fibrous tissue encapsulating the tubercles.
2. Caseation and calcification of the tubercles or tubercular debris, thus causing cavities to dry up (natural cure). Remember, however, the disease, having become chronic, may take on a rapid form through some inflammatory condition elsewhere.

Special Symptoms.—

(1) *Hæmoptysis.*—The blood is bright and frothy in the early stage, but later it may be dark from stagnation or venous congestion. During the excavation stage small aneurismal swellings on unsupported blood-vessels *may burst and cause speedy death*. Often slight hæmoptysis is the first symptom of phthisis.

(2) *Cough* is a constant symptom; at first slight, then gradually becoming hacking, paroxysmal, and painful.

(3) *Expectoration* at first is muco-purulent, but later becomes more purulent and copious. It is non-aerated, often blood-stained, and shows the peculiar coin-like arrangement when expectorated into water. Often little grey hard pellets are present, with abundance of elastic tissue and bacilli.

(4) *Pyrexia.*—At first the temperature is elevated only in the evening, but towards the end the temperature may oscillate

continually between 100° and 104° F. Such a temperature is highly suspicious of sepsis due to absorption of broken-down tissue. It is usually accompanied by the "hectic" flush, dilated pupils, and severe night-sweats. When the *so-called* night-sweats occur in the *earlier* stages, they are probably due to reflex vaso-motor disturbances *and not sepsis*.

(5) *Loss of Flesh*.—This is *generally most marked*, especially in the later stages, and where pyrexia is prominent. There is often extreme muscular irritability (myoidema).

(6) *General Appearance*.—The chest is frequently flattened, the antero-posterior diameter being short. The ribs present marked obliquity, so that the epigastric angle is acute. The angles of the scapulæ may stand out prominently from the ribs (alar chest). In the late stages there are marked clubbing of the finger-tips and curvature of the nails.

Complications.—

1. *Pleurisy* is most commonly present, but it is doubtful whether it should be looked upon as a complication. The late Sir William Gairdner in his excellent monograph says: "It is not to be regarded as a fatal complication, but as a healing power, inasmuch as it prevents perforation of the pleura, or even obliterates the cavity between the two layers, thus preventing empyema, pneumothorax, etc. *It is a curious and beautifully conservative arrangement* that in most cases the pleuritic adhesions are often *in advance* of the actual deposit of tubercle near the surface, and still more in advance of its softening."

2. Pyothorax.

3. Pneumothorax.

4. Spreading of the tubercular disease to the *larynx*, meninges, peritoneum, etc.

5. *Diarrhœa*.—This is usually a late symptom. It may be due to mere intestinal catarrh, to tuberculous ulceration of the bowel, or to amyloid disease. The two latter conditions are very intractable.

6. Tuberculous ulceration of the bowel may lead to *hemorrhage*, or more rarely to *perforative peritonitis*.

Physical Signs.—We have already pointed out that the typical signs given in the table are often absent or masked by various conditions. It is often difficult to say whether a cavity exists or not, owing to—

1. Its small size.
2. Its being deep down, and more or less healthy lung intervening between it and the surface.
3. Thickened pleura.
4. Compensatory emphysema.
5. Breath-sounds being drowned by extensive bronchitis.
6. Blocking of the tubes leading to the cavity.
7. Irregularity of its walls.
8. Too high or too low tension in its walls.

The “cracked-pot sound” is not absolutely diagnostic; it may be heard in crying children with healthy lungs, and in some cases of pneumonia. We thus see there are many *pros* and *cons* to be considered before diagnosing a cavity, and unless the *general condition and history of the patient* are well considered, physical signs will often lead to an erroneous diagnosis. Post-mortem examinations prove this to be so.

Treatment will be considered under four heads—specific, hygienic, dietetic, and medicinal.

Specific Treatment.—It has already been stated (see p. 272) that treatment by vaccines is at present chiefly confined to localised staphylococcic and tuberculous infections. Where it is possible to convert a systemic infection (pyrexial phthisis) into a localised one, as by prolonged rest in bed, milk diet, subjugation of fever, etc., then inoculations of *tuberculin*, which are to be determined both as to their dose and their frequency by estimation of the opsonic index, may be used with marked benefit, and may possibly lead to cure.

Other forms of specific treatment, as intravenous or intra-

laryngeal injections of antiseptic drugs, have not fulfilled the expectations formed of them.

Tuberculin is of great use as a means of diagnosis in doubtful cases.

Hygiene.—Patient must wear wool or flannel next the skin, take gentle outdoor exercise, and sleep *alone* in an airy bedroom, the windows being kept open day and night. He should be well rubbed after tepid baths, and absolute cleanliness observed as regards the teeth and mouth. The expectoration should be received into an antiseptic solution, and never allowed to dry on the carpet, etc. Whilst the patient should be kept warm, do not overload him with heavy clothes—do not, in fact, coddle him.

Dietetic.—We should aim at giving our patient *as much food as he can possibly assimilate*. Alcohol may be given where there is much debility, or if the appetite is impaired. It is not necessary as a routine measure. The patient's great dislike to fats of all kinds hampers dietetic treatment a great deal. Koumiss and peptonised meats must always be remembered when the assimilative powers are weak.

Medicinal.—

1. *General.*—Tonics, such as hypophosphites, maltine, and cod-liver oil, are the principal remedies prescribed.

2. *Symptomatic.*—Any grave complication must be treated energetically.

(1) The cough.—As this is a persistent and constant feature of the disease, avoid rushing to the usual cough mixtures at once. A most common exciting cause of the nightly cough is the changing from a warm room to a cold bedroom; or again, tickling of the fauces by the uvula. A very good cough mixture is—

℞ Tinct. Chloroformi et Morphinæ . . . ʒij.
 Acid. Nitric. Dil. ʒij.
 Glycerinum ad ʒiiss.
 Fiat mist. ʒi when the cough is very troublesome.

Inhalations "*moist or dry*" are of the utmost value.

- (2) The *night-sweats*.—Picrotoxin, quinine, atropine, and oxide of zinc are the favourite remedies.
- (3) The *diarrhoea*.—It is usually best controlled by mineral astringents, in combination with opium.
- (4) *Hæmoptysis*.—Absolute rest and cold applications. Tinctura ferri, hamamelis, gallic acid, ergotine, or *best of all a full dose of opium*.

Other complications must be treated on general principles.

Change of Air.—Under this heading we shall include—

1. Long sea voyages.
2. Change of residence.

Sea voyages undoubtedly do good in many cases of early phthisis. Whether the benefit is due to a general bracing up of the nervous and muscular systems, or whether the sea air (containing as it does, iodine, bromine, etc.), exerts a beneficial *local* influence, is not yet determined, but probably both factors contribute to the cure. The comparative sterility of sea air undoubtedly contributes to the result. Before deciding to send a patient on a long voyage several points must be considered. Is the patient a good sailor? If *not*, is the severe sea-sickness likely to produce hæmoptysis, or prevent proper feeding? Is the patient well off and likely to receive every comfort on board? Remember a woman cannot “rough things” as easily as a man; and further, many things which are not *inconvenient* to a man, must be so, to a woman. No patient should go without a relative or friend accompanying him. Lastly, sea voyages are contra-indicated in the later stages of phthisis.

Change of Residence.—A phthisical patient requires either *dry cold* or warm air. In the earlier stages cold dry air is the best. New Mexico, Davos, and Canada are the best examples of such residences. For warm climates we might select New Zealand, Madeira, Algiers, Torquay, Hastings, etc. Remember it is absolutely cruel quackery to send a patient far advanced in phthisis away from home and friends, only too frequently to die amongst strangers.

Open-Air Treatment.—This is now extensively adopted.

Its leading principles are that a consumptive should pass as much of his life as possible in the open air, remote from the germ-laden atmosphere of towns, but not necessarily out of his own country, and that he should eat as much as possible, even beyond his appetite. For this purpose sanatoria have been and are being built in suitable spots in Scotland and other countries. Open-air exercise is insisted on in suitable cases. Those unable to take it lie warmly wrapped up on verandahs freely exposed to the atmosphere during the day, but sheltered from rain or snow. Windows in the wards are always open, and patients are thus practically outside for the whole twenty-four hours.

Much benefit has been derived from this treatment, especially in early cases; but it is at present too costly for the poorer classes, for whom only a few sanatoria are as yet available. In their case the physician can often do no more than see that all sputa are destroyed, for the protection of others, and that free ventilation is carried out as far as may be, coupled, of course, with the usual dietetic and medicinal remedies. Vigorous efforts are being made by many public bodies to make it punishable to spit upon the street or in public vehicles. If this can be universally carried out, there is no doubt that the mortality from phthisis will be substantially diminished.

In some cases aspiration of the cavities, followed by injections of antiseptics, has proved of use.

Laryngeal Phthisis has been considered under diseases of the larynx.

CHRONIC OR INTERSTITIAL PNEUMONIA.

(CIRRHOSIS OR FIBROSIS OF LUNG.)

By chronic or interstitial pneumonia is meant an induration of the lung tissue, independent of tuberculous induration (fibroid phthisis), characterised by overgrowth of the fibrous elements, due to the operation of slowly acting irritants, and attended clinically with symptoms of pulmonary embarrass-

ment; later on, the symptoms are *dependent on grave structural change and breaking down of lung substance.*

Causes.—

1. Inhalation of dust particles (pneumonokoniosis). Examples, anthracosis (coal miner's lung); silicosis (mason's lung).
2. Irritants in the form of animal poisons.—Glanders, syphilis (white hepatisation).
3. Excessive use of alcohol.
4. Result of acute pneumonia, pleurisy, etc.

Pathology.—The morbid anatomical changes depend on the cause, and so the reader is referred to a text-book on pathology. We shall merely state here the conditions common to all.

1. Great overgrowth of interstitial connective tissue, encroaching upon and destroying the air vesicles.

2. Changes in the bronchial tubes; their muscular coats are replaced by fibrous tissue, which subsequently yield to intra-pulmonary pressure, and form bronchiectatic cavities (see Bronchiectasis). Sometimes their lumen is occupied by caseous masses, and very frequently *obliterative* changes take place, and the lumen becomes totally obliterated.

3. *Obliterative changes in the pulmonary and bronchial blood-vessels leading to severe systemic and local changes.*

Symptoms.—A careful digest of the outstanding features of the morbid structural changes ought to enable us to anticipate the more constant symptoms. The disease runs a very chronic course, and is usually unilateral. Cough may be the only symptom complained of for a long time. Later, the symptoms are those of bronchiectasis.

Physical Signs.—The affected side is retracted, much flattened, and the intercostal spaces more or less obliterated. The percussion note will depend on the state of the bronchi, ranging from *absolute dulness to a boxy note*; indeed, in the later stages, the physical signs may be those of small cavities with extensive bronchitis. The retraction may lead to compensatory emphysema of the opposite side, dragging of the

heart out of position (often upwards and to the left), dragging on the great vessels, etc.

Treatment.—Remove if possible the exciting cause, and then treat on general principles already laid down, in speaking of the various pulmonary affections.

PNEUMOTHORAX.

Pneumothorax means the presence of air in the pleural cavity.

Causes.—

1. Traumatism, such as punctured wounds, laceration from the end of a broken rib.

2. Causes from the lung side—

- (1) Bursting of a pulmonary abscess into the pleura.
- (2) Gangrene of the lung.
- (3) Excessive intra-pulmonary pressure.
- (4) Tubercles breaking down, causing undermining and perforation of the pleura when adhesions have not been set up.

Physical Signs.—

Inspection.—Bulging may be local or general on the affected side.

Percussion.—Usually yields a hyper-resonant note. If the valve-like opening be patent, a typical crack-pot sound can be elicited.

Palpation.—Absence of vocal fremitus.

Auscultation.—Absence of breath-sounds. On coughing the sounds produced are peculiarly amphoric and have a metallic ring. If the chest be struck with two coins whilst listening with the stethoscope the characteristic bell or anvil sound is heard.

The admission of air into the pleural cavity implies the admission of organisms. Inflammatory changes are thus set

up, and an effusion, either sero-fibrinous (*hydropneumothorax*) or purulent (*pyopneumothorax*), is the consequence.

Hydropneumothorax.—A condition where air is *present above* and fluid *below* in the pleural cavity.

Physical Signs.—*In the upper part*—those signs already described as diagnostic of pneumothorax, plus *hippocratic succussion* (the term applied to the splashing sound heard on shaking the patient).

Below—the signs diagnostic of fluid already discussed under pleurisy.

Pyopneumothorax means the presence of air and pus in the pleural cavity. The physical signs are the same as those of hydropneumothorax.

Symptoms.—The symptoms of pneumothorax depend largely upon the manner in which it was produced, and the quantity of air present. They may be summed up as follows:—

1. Sudden pain at time of rupture with or without collapse.
2. Great dyspnœa.
3. Quick and small pulse.
4. Shallow and rapid breathing.

In many cases of advanced tuberculosis, the symptoms are not very urgent, and dyspnœa is not marked.

Treatment.—Subdue the pain by a hypodermic injection of morphine, hot poultices, diffusible stimulants, etc. Aspiration is sometimes of value, if the air is confined in the pleura under high pressure. Pyopneumothorax may be treated by incision if the lung is only slightly affected. Where there is advanced pulmonary disease, incision is not indicated.

HYDROTHORAX.

Hydrothorax—*i.e.*, dropsy of the pleuræ, is a term used to denote fluid in the pleural cavity, the result of a passive process, as seen in Bright's disease, etc.

The main points of difference between it and real pleuritic effusion are—

1. It is not preceded by acute symptoms of inflammation, therefore, absence of friction.
2. Usually bilateral.
3. The fluid gravitates with movements of the patient to a far greater extent (like ascites).
4. Aspiration is not likely to do more than *temporarily relieve pressure*.
5. Associated with signs of dropsy elsewhere.
6. Absence of pain, but dyspnoea is greater than in acute pleurisy.

TUMOURS OF THE LUNGS.

CANCER.

Primary cancer of the lung is very rare. The secondary variety occurs in the form of either *dense*, hard, irregular nodules, or soft (medullary) masses, scattered throughout the lung tissue. The original sources are mostly—

1. Cancer of the bronchial glands, which spreads inwards at the root of the lung, or
2. Cancer of the liver, which invades the diaphragm, and finally the pleura and lung.

Symptoms are insidious and varied. The amount of consolidation often causes pneumonic symptoms. The ulcerative process gives similar signs to phthisis; and hæmoptysis is generally present.

Diagnosis.—When there are symptoms of malignant mischief elsewhere, *marked pulmonary symptoms point to the invasion of the lung*. Pleuritic effusion may be so well simulated that puncture is sometimes required to clear up the diagnosis. The disease is usually fatal in about six months from the onset of symptoms.

Other tumours are too rare to need a detailed account in a work like this.

DISEASES OF THE KIDNEY.

ALBUMINURIA.

By albuminuria is meant the presence of serum-albumin and serum-globulin in the urine. As albumin is one of the essential constituents of the blood, an escape of it in the urine must necessarily be looked upon with suspicion, if not with anxiety. Formerly, all cases of albuminuria were regarded as varieties of Bright's disease, but it has been conclusively shown that albuminuria is sometimes present in (as far as we can make out) healthy individuals. However, "*a persistent albuminuria, especially if the albumin tends to increase, must always be looked upon as a grave and significant condition.*" In other words, just as palpitation of the heart may exist without cardiac disease, it may on the other hand be a prominent symptom of a grave organic lesion; so with albuminuria, it may or may not be a serious omen.

Albuminuria may be either of renal origin, or dependent upon some lesion of the urinary tract below the kidney. The cause of renal or essential albuminuria is probably to be found in the glomeruli. It is held that the epithelial cells of the glomerular tuft exert a selective influence in excretion, inasmuch as they allow the water and certain soluble salts to pass, but prevent albumin from so doing.

It has also been maintained that albumin *normally* escapes through the glomeruli into the tubules, but is rapidly taken up again by the cells of the tubules, and *reabsorbed into the blood*. This is doubtful, although the cells of the convoluted

tubules certainly have a decided *selective function*, as shown in the excretion of urea, etc.

The first theory would demand for the production of albuminuria a diseased condition of, or at least some alteration in, the epithelium of the glomeruli.

The second theory demands "increased blood-pressure," in order to explain the escape. A great increase of the renal blood-pressure injures the epithelium of the tubules. Possibly both conditions play their parts, but the glomerular theory is certainly the more satisfactory.

Experimentally, we may produce albuminuria by—

1. Pressure upon (not closure of) the renal veins; the pressure in the glomeruli is increased thereby.

2. Closure of the renal artery, and subsequent re-establishment of the circulation; this interferes with the nutrition of the renal cells.

3. Ligature of the aorta *below one* kidney, and extirpation of the other.

4. Ligature of the aorta above the renal arteries.

5. Compression of the trachea; this leads to asphyxia, and consequent rise of blood-pressure. (HALLIBURTON.)

Clinically, we get albuminuria—

1. Due to—

Morbid conditions of the kidney, such as acute and chronic inflammation, waxy degeneration, renal calculi, tumours, etc.

Diseases of the urinary apparatus below the kidney
Hæmic changes—anæmia, leucocythæmia, etc.

Certain fevers, especially scarlet fever and diphtheria.

Pregnancy.

Certain poisons—cantharides, turpentine, arsenic, phosphorus, excessive use of morphia, etc.

Venous congestion, consequent on hepatic, pulmonary, or cardiac diseases, etc.

2. After certain diets, especially in those who pass much oxalates in their urine (oxaluria).

3. In a *remittent or cyclic form*, occurring in apparently healthy people; albumin is in such cases only present in the urine at certain periods of the day, as after rising, after meals, or following severe exercise or mental emotion.

Treatment of Albuminuria.—No condition requires more careful treatment. The first thing to do is to settle if possible *whether it is functional, or the result of structural disease of the kidney or urinary passages*. This can only be done by careful consideration, and *frequent* examinations of the urine. Do not forget that albumin is *temporarily* absent in many grave cases of renal disease. The following are the chief indications for treatment.—

1. Remove if possible any obvious cause.
2. Prescribe a non-nitrogenous diet as far as possible.
3. Avoidance of all substances (food or drugs) likely to increase unduly the blood-pressure, or cause irritation of the renal tubules.

Further treatment will be discussed under Nephritis. For tests for albumin see Examination of the Urine.

BLOOD IN THE URINE.

Blood in the urine is another very grave symptom. Two forms are described—

1. Hæmaturia, or bloody urine proper.
2. Hæmoglobinuria, a condition marked by the presence of blood-pigment in the urine, but *few or no* blood corpuscles. *The blood pigment is generally methæmoglobin* (chocolate colour).

Causes of Hæmaturia.—

Diseases of the kidneys and urinary apparatus below them.

Extensive bruises (absorption of blood pigment).

Scurvy (especially in children).

Malignant forms of fevers, especially the malarial type.

Traumatisms.

No discoverable cause (Gull's "epistaxis of the kidney").

Drugs which directly irritate the kidney,—turpentine, cantharides, carbolic acid, etc.

Parasites, especially the bilharzia hæmatobia.

Diagnosis.—

1. *By the colour of the urine*, which ranges from a slight smoky tint to deep red or even porter colour.

2. *Microscope*.—Detection of blood discs (in fresh urines).

3. *Spectroscope*.—The characteristic bands of hæmoglobin (generally the reduced Hb).

4. Ozonic ether and guaiacum gives a blue tint. The reaction is also given by nasal mucus, saliva, and iodine or the iodides.

If the blood comes from above the ureters, it is usually freely mixed with the urine.

If from the ureters, blood may be clotted in the form of moulds.

If from the bladder, is either mixed, or appears in greatest quantity towards the end of micturition.

If from the urethra, before or during the *first* part of micturition.

There are no definite means of ascertaining the site of the hæmorrhage by mere examination of the urine alone.

Treatment varies with the cause. Rest in the recumbent posture. Full doses of opium (contra-indicated in organic renal disease). Ergot.—Lead Acetate.—Hazeline.—Hydrastis Canad.

Parasitic Variety.—Injections into the bladder of iodide of potassium (5 grains to 3i). Santonine, or male fern, internally.

Causes of Hæmoglobinuria.—

1. *Toxic Form*.—Due to the action of certain poisons, such as *nitrites*, arseniuretted hydrogen, carbon monoxide, bile, etc.

2. *Paroxysmal Form*.—This condition is characterised by the occasional passage of bloody urine, the pigments *only* being present. The causes are not known, but there seems to be a great causal relation between this condition and disturbances of the vasomotor system; so much so that it has been termed

“Raynaud’s” disease of the kidneys. It bears also a close relationship to syphilis. The most commonly associated conditions are—

- (1) Vasomotor disturbances.
- (2) Increased hæmolytic action of the liver and spleen.
- (3) Extensive superficial burns.

(I saw three such cases in a colliery practice.)

In newly born infants hæmoglobinuria may accompany the usual destruction of red corpuscles.

Symptoms.—The attack may come on after the morning bath, mental exhaustion, or exposure to cold. There is usually pain in the lumbar region during the attack. Vomiting, anorexia, and jaundice are sometimes present.

Treatment.—Fresh air and tonics (especially quinine). During the attack keep the patient *warm* and give hot drinks.

URÆMIA

Is the name given to the symptoms that arise from retention within the blood of certain excrementitious substances normally excreted by the kidneys. It is usually associated with inflammatory affections of the kidneys and suppression of, or diminished quantity of urine. Numerous theories have been advanced to explain the condition of toxæmia which is thus set up, but none of these is free from objection. The most important are—

1. The symptoms are due to retention in the blood of excess of urea. To this it is objected that the blood does not always contain excess of urea, that urea may be injected into the blood without causing uræmia, that anuria may exist without it, and that it may occur in spite of a normal elimination of urea.

2. The urea is decomposed in the blood into carbonate of ammonia, which causes the symptoms. But carbonate of ammonia may not be present in uræmic blood, and the odour of it in the breath is due to decomposition in the mouth, not in the blood.

3. The symptoms are due not to urea alone, but to the total of the normal excreta. None of these except the inorganic salts is present in sufficient quantity to be toxic.

4. The symptoms are due partly to the salts of potassium, partly to intermediate products of proteid waste which resemble alkaloids. Bouchard regards the condition as depending partly upon absorption from the intestine (constipation is very constant in nephritis), partly upon non-elimination by the kidneys.

Evidently the pathology is as yet unsettled, and probably no one theory is adequate to explain all the cases.

Uræmia may be *acute* or *chronic*. The acute form presents symptoms of a cerebral type; in the chronic form the symptoms are both cerebral and digestive.

Acute Uræmia: Symptoms.—*Epileptiform convulsions*, which may come on suddenly or be preceded by headache, vertigo, or nausea. They are usually bilateral, but may be unilateral. There may be a single fit, followed by stupor, or a series of fits, with intervening coma (*status epilepticus*). Tonic spasms or tetany, muscular tremors or twitchings may replace convulsions.

Coma accompanies the convulsions, but may occur independently.

The seizure may be *apoplectiform*.

Delirium or even *acute mania* may occur.

Amaurosis may accompany the convulsions, or occur independently, with no other symptom than headache. The patient finds himself suddenly totally blind in both eyes. There are no changes in the fundus oculi, and sight is perfectly recovered in a few hours or days.

Chronic Uræmia: Symptoms.—(A) Cerebral. Persistent *headache*, *vertigo*, and *drowsiness*, either continuous or intermittent, passing into a condition of apathy or stupor, and ultimately into the *typhoid state*. There may be repeated attacks of *dyspnœa*, with rapid shallow breathing and cyanosis, or *Cheyne-Stokes breathing* may occur. *Pruritus* is not uncommon.

(B) Digestive. *Vomiting*, at first in the morning or after meals, later independent of food. The vomitus may contain urea. *Diarrhœa* is frequent. It may be simply catarrhal, or due to ulcerative colitis. *Hiccough* may be troublesome.

Prognosis.—The first attack of uræmia may prove fatal, but in acute nephritis complete recovery is frequent. In chronic nephritis the patient may survive the attack for several years.

Diagnosis.—If a patient is first seen in the comatose state, apoplexy, alcoholic poisoning, and opium poisoning must be excluded. The urine, removed by catheter, must be tested for albumin, the pulse examined for evidence of arteriosclerosis, and the heart for hypertrophy of the left ventricle. Conjugate deviation of the eyes speaks for cerebral hæmorrhage. In opium poisoning the pupils are contracted, in alcoholic coma usually dilated, in uræmia they are variable, and may be unequal. Remember (1) that cerebral hæmorrhage is common in cirrhosis of the kidney, and (2) that though a patient's breath smells strongly of alcohol, he may yet have uræmic, and not alcoholic, coma. If there be any doubt, treat the case as if it were the graver lesion.

ACUTE BRIGHT'S DISEASE.

(ACUTE PARENCHYMATOUS NEPHRITIS.)

“By Bright's disease we mean a non-suppurative inflammation of the kidneys. To understand the classifications that have been made it is necessary to recognise the following structures in the kidney, which are liable to diseases, *more or less independent of one another*. They are—

“1. The *Tubules* with their epithelium, forming the parenchyma of the kidney.

“2. The *Interstitial tissue*, very small in quantity in the healthy organ, but liable to considerable increase by inflammatory processes.

“3. The *Blood-vessels* and the *glomeruli*; consisting of the vascular tufts, the capsule, and the epithelial cells covering the former and lining the latter.” (Dr. Taylor's *Practice of Medicine*.)

Obviously in a nephritis the inflammatory changes may be most marked in the *tubules*, or in the *interstitial tissue*, or in the *blood-vessels*; but it must be equally clear that no *one* of these special tissues can be affected to any great extent *without implicating the other components of the kidney structure*. In other words, though the various types of Bright's disease are based upon the variety of tissue principally involved, still, it must be distinctly understood that *the kidney structure as a whole shares in the inflammatory changes*. This applies with particular emphasis in acute cases.

Acute inflammation of the kidneys is a disease characterised by febrile disturbances, grave changes in the urine, and dropsy.

Causes.—

1. Exposure to cold, especially when the body is overheated.

2. Acute specific fevers and septic states, especially scarlet fever and ulcerative endocarditis. (Albuminuria without the other symptoms of acute nephritis is common in all fevers.)

3. Certain irritating drugs, *e.g.*—cantharides, copaiba, turpentine, etc.

4. Extensive burns involving the abdomen. The nephritis is due to septic absorption.

5. The disease sometimes occurs in pregnancy.

Males are somewhat oftener attacked than females, and acute nephritis is commoner before the age of forty than later.

Pathology.—Two types are usually distinguished,—*i.e.*, the ordinary or catarrhal form, and the so-called infective or glomerulo-nephritis.

1. *Catarrhal Nephritis*.—The kidneys are large, much injected, and the cortex is seen to be disproportionately enlarged. On section, the cut surfaces show up the Malpighian bodies as

deep red points ; here and there are patches of extravasated blood. The tubules show marked changes, especially the convoluted portions, the epithelium undergoes cloudy swelling, followed by proliferation and detachment of the cells, which form casts or masses of granular fatty debris, often to the extent of blocking up the tubules. It must not be forgotten, however, that though the tubules are the parts principally involved, the interstitial portion also takes part in the inflammatory process.

2. *Glomerulo-Nephritis*.—In this form the glomeruli principally suffer, and it is usually the result of infective fevers, especially scarlatina. After a preliminary engorgement of the blood-vessels, extensive leucocyte emigration takes place, filling the glomerular capsule, the capillary vessels often burst, and the blood flows into the tubules. The epithelium of Bowman's capsules often proliferates to such an extent that the tufts become obliterated by the pressure, and in time the latter may be actually changed into fibrous nodules. Of course the tubules and interstitial tissue are also involved, though not to the same extent.

Symptoms are, at first, chilliness, pain in the back, vomiting and pain over the brows ; the temperature is raised (but may never be high), the characteristic œdema rapidly appears, at first in the conjunctivæ, eyelids, and cheeks, but later it becomes general. The pulse is usually quick and of *high tension* from the beginning. The urine is voided frequently, but scant in quantity, and indeed may be suppressed for a time. It is dark from the presence of blood, exhibits the well-known smoky hue, and generally gives a copious deposit of urates, blood discs, granular debris, epithelial, fatty, and hyaline casts, and *a large amount of albumin*. Though the urine is of high specific gravity, the total amount of urea passed daily is much decreased. As the disease advances, the anasarca becomes more marked ; there may be dangerous dropsy of the serous sacs, œdema of the base of the lung or of the glottis. The bowels become constipated, the tongue dry,

and there is great thirst. Hypertrophy of the heart is seldom marked in acute cases, although acute dilatation of the heart may occur. If improvement does not quickly take place, grave dangers arise from the accumulation of excrementitious material in the blood; anæmia becomes pronounced, and intense headache, convulsions, and coma often usher in a fatal uræmia. Most cases, however, with care, recover; others pass into the subacute stage or "large white kidney."

The more important complications to remember are—

1. Excessive dilatation of the heart in debilitated patients.
2. Edema of the lungs or glottis, hydropericardium or hydrothorax.
3. Albuminuric retinitis may be present where the acute attack is an exacerbation of pre-existent chronic disease.
4. Uræmia.

Treatment.—Absolute rest in bed, patient to be laid between blankets; hot poultices (not blisters), or dry cupping over the loins, a free saline purge, diluents in an effervescing form, and a *non-nitrogenous* diet, form the routine treatment. The diet should if possible be of milk only. Remember that urates are highly irritating to an inflamed kidney, *hence the necessity of diminishing the quantity of proteids taken*, and the indication for the use of these drugs which render the urates more soluble. Acetate of potash and nitrous ether with hyoscyamus, form a splendid mixture, as we have at once *a solvent of urates, a dilator of the peripheral arterioles, and a sedative to the urinary tract*. If there be excessive renal congestion causing marked diminution or suppression of urine, diuretics should not be given till the congestion is relieved by cupping, hot air-baths, or leeching. Complications must be treated as they appear.

Excessive Dropsy.—Hot air-baths, pilocarpine, compound jalap powder, claterium, Southey's tubes, tapping, etc.

Uremia.—Same as for excessive dropsy *plus* chloroform during the convulsions, and wet cupping, or even venesection.

Severe Dyspnœa.—Nitrite of amyl is of great service.

CHRONIC BRIGHT'S DISEASE.

Chronic Bright's disease may be divided into two forms—

1. The chronic parenchymatous or tubal nephritis (large white kidney), leading in the later stages to contraction (small white kidney).
2. Chronic interstitial nephritis (small red or gouty kidney, cirrhosis of the kidney, granular kidney).

Chronic parenchymatous or tubal nephritis may either follow an attack of acute nephritis or arise independently. It is a disease of comparatively early adult life. Chronic interstitial nephritis is rare before the age of forty.

Lardaceous or waxy disease of the kidney (pale waxy kidney), although often described as a form of Bright's disease, is really a degeneration, and not at all of an inflammatory nature. But it is often *accompanied* by interstitial nephritis.

Before describing these varieties in detail, we must consider some *general* facts in reference to renal disease. Now suppose we get defective renal secretion, what must happen?

1. Retention of waste and poisonous products in the blood.
2. Damage to the vascular walls by those poisonous products.
3. Anæmia; and consequently all the troubles produced through imperfect nutrition of *all the tissues and organs in the body*. One of the chief of these is *wasting*, often masked by accompanying œdema.

We must expect and therefore describe changes under six headings—

1. *Changes in the kidney structure—*

Interstitial tissue.

Tubules and their epithelium.

Blood-vessels.

2. *Changes in the urine*—as regards quantity and quality—*i.e.*, the presence of pathological constituents—

Albumin.

Blood.

Casts, which may be granular, fatty, or hyaline.

Defective excretion of urea.

3. *Changes in the circulatory system*—

The heart is often enormously hypertrophied and dilated.

The arteries are sclerosed and thickened.

The pulse is characteristic—it is peculiarly hard, and of high tension, the dicrotic wave being almost obliterated.

[The cause of the hypertrophy and the nature of the arterio-sclerosis are still unsettled. As the high tension occurs quite early in even *acute* Bright's disease, increased peripheral tension set up by irritation of the vascular walls is the probable cause, and this would account for subsequent hypertrophy of the heart. Cohnheim considered that the fact of more force being required to drive the blood through a *diseased* kidney, would explain the cardiac hypertrophy. The question, however, is still *sub judice*.]

4. *Ocular changes*, consisting of—

Œdema of the retina, causing opacity and swelling.

White glistening patches due to fatty degeneration of

Müller's fibres, etc.

Flame-shaped extravasations of blood.

More or less optic neuritis.

Diffuse retinitis albuminurica.

5. *Dropsy* first appears as œdema of the face (affecting the eyelids first) and of the feet: later, general anasarca, and dropsy of the serous cavities—*i.e.*, ascites, hydrothorax, or immense dropsy of the scrotum, may all occur. The position of the fluid is influenced by gravitation.

The dropsy is at first most apparent in the morning. Many factors contribute to the causation of renal dropsy—

- (1) The anæmic and hydræmic state of the blood.
- (2) Degeneration of the vascular walls.
- (3) Déficient secretion of urine.
- (4) Imperfect cardiac action and pulmonary engorgement (late).

6. *Tendency to Uræmia*.—Uræmia has already been discussed fully.

There is no doubt that Professor Grainger Stewart struck the keynote to the proper understanding of the various clinical phenomena, when he pointed out "that chronic inflammation of the kidney differed in no way from that of other organs; that, given a slowly progressing inflammatory action, we should expect at first a stage of enlargement, and finally a state of atrophy."

Chronic inflammation may or *may not* be preceded by an acute attack; and lastly, when the condition begins as an *acute* nephritis, before it can become chronic, *there must be an intermediate or subacute stage*.

I would strongly urge the junior student to digest the foregoing remarks, and especially remember the relations between the liver and kidneys. If he does this, there can be no difficulty in understanding a most important section of practical medicine.

Chronic parenchymatous nephritis is frequently a sequel of an acute attack. It leads either to fatty degeneration of the kidney substance ("large white kidney"), in which the capsule is non-adherent, the cortex yellowish or mottled, and the pyramids injected, or, later, to fibrous degeneration ("small white kidney"). In this stage the capsule is adherent, the surface granular, the cortex narrow and pale. *Chronic interstitial nephritis* is often associated with gout, chronic

alcoholism, or excessively nitrogenous diet, associated with the less active metabolism of middle age. The kidney is small, the capsule firmly adherent, the surface purplish red, uneven, studded with small cysts, the cortex narrow. Fibrosis is very marked, and arteriosclerosis is pronounced.

In the early stages of chronic parenchymatous nephritis ("subacute nephritis") the *symptoms* approximate to those of the acute form. The urine is scanty, highly albuminous, contains blood, and is defective in urea. Fatty casts are present in quantity. Dropsy is prominent (Fagge quotes the saying that "large white kidneys make a large white body"), and affects the internal cavities even more frequently than in acute nephritis. In the later stages the urine is increased in quantity, blood and albumin are less, the casts tend to become granular or hyaline, and dropsy is less prominent. Cardio-vascular changes are more pronounced.

The onset of chronic interstitial nephritis is most insidious. Often there is nothing to attract the patient's attention to such a serious condition, beyond languor, a desire to micturate more frequently, possibly dyspeptic symptoms, diffuse headache, and slight stiffness of the eyes in the morning. Albuminuria may be absent for a considerable time. When the disease is well marked, and cardiac hypertrophy begins to fail, the symptoms become very pronounced, such as marked pallor, breathlessness, attacks of uræmic asthma, dimness of vision, and dropsy of the cardiac type. It is very essential that the condition should be early diagnosed, and careful examination of the pulse, retina, and urine ought to go far to make an accurate as well as an early diagnosis. The foregoing remarks, along with the diagnostic table which follows, should enable the beginner to have a clear idea of the elementary facts of Bright's disease. A more exhaustive account cannot be given here, but after the final examination is passed, no practitioner should lose time before perusing one of the many valuable monographs on the subject.

Treatment of chronic Bright's disease must be considered under two heads—

1. General treatment.
2. Symptomatic treatment.

General Treatment.—

Our indications under the first heading are to put the patient under the most favourable hygienic conditions. In the stage of large white kidney the indications for treatment are obviously much the same as in acute nephritis. Later, exercise in the open air, and woollen or flannel underclothing, are imperative. Diet is no less important; therefore food which contains the *most* nutritive properties with the *least* nitrogenous compounds must be prescribed. No rigid diet can be laid down for all cases, each must be treated on its own merits; but in all cases, beer, heavy wines, and excessive consumption of sugar should be condemned.

Medicinal Treatment.—Keep the bowels open by the use of saline purges. Improve the vascular tone by the administration of strychnine and iron. Cardiac tonics are necessary when the heart begins to fail. If vascular tension is very high, digitalis should be combined with some vaso-dilator such as spt. æth. nitros., or nitroglycerine.

Though it is generally held that mercurials are badly borne in renal affections, there can be no doubt as to the efficacy of small doses of pil. hydrarg. or calomel, given at night-time, and followed by a tumblerful of Friedrichshall water in the morning. Hepatic depletion by brisk cathartics is often more efficacious than any other treatment in allaying urgent symptoms.

Symptomatic Treatment.—

Headache may be relieved by a mixture containing pot. iodid., digitalis, and caffeine. When the tension is very high, a good purge and a few whiffs of nitrite of amyl often act like a charm.

Dropsy.—When excessive, may demand removal of the fluid by Southey's tubes. A saturated solution of mag. sulph., administered in the morning, often removes enormous quantities of water.

Albuminuria.—If the escape of albumin be excessive, ergot or gallic acid may be tried. Drugs, however, have very little effect in controlling the loss of albumin.

Differential Diagnosis of the Forms of Bright's Disease.

CHRONIC NEPHRITIS.					
Etiology.	ACUTE NEPHRITIS.		CHRONIC PARENCHYMATOUS NEPHRITIS : LARGE WHITE KIDNEY.	SECONDARY CONTRACTED KIDNEY : SMALL WHITE KIDNEY.	CHRONIC INTERSTITIAL NEPHRITIS : PRIMARY CONTRACTED OR SMALL RED KIDNEY : CIRRHOTIC OR GRANULAR KIDNEY.
	Chill ; acute poisoning (cantharides, etc.) ; acute infections (scarlatina, etc.).		Acute nephritis ; prolonged influence of cold and damp ; malaria, cardiac lesions, phthisis.		
	Quantity.	Scanty.	Rather less than normal.	Not less than normal ; usually increased.	Very abundant.
	Colour.	Turbid, pale red or smoky to deep red.	Turbid, resembling meat infusion.	Fairly clear.	Clear, pale.
	Specific gravity.	High.	Somewhat raised, may be normal.	A little below normal.	Low.
Characters of Urine.	Blood.	Abundant.	Commonly present.	In small quantity.	Usually absent.
	Albumin.	Abundant.	Abundant.	In moderate quantity.	In very small quantity ; may be absent for some time.

Sediment.	Abundant. White and red corpuscles; blood-casts, epithelial and granular casts; urates.	Abundant. White and red corpuscles; numerous casts, especially <i>jetty</i> .	In moderate quantity. Casts fairly numerous, granular and hyaline chiefly.	Very scanty. Casts few, chiefly hyaline
Salts and Urea.	Marked diminution of urea, chlorides and phosphates.	Diminution of urea, etc.	Marked diminution of urea, etc.	Marked diminution of urea, etc.
Cardiac Hypertrophy.	Usually <i>absent</i> , unless in acute exacerbations of chronic disease.	Sometimes present.	Usually present.	Almost always present, and very considerable.
Prosy.	Marked; shifting from place to place with position of patient.	Marked; <i>dropsy of internal cavities</i> .	Moderate; both subcutaneous and internal.	Usually absent; later dropsy of cardiac type when heart fails.
Uremia.	Frequent.	Fairly frequent.	Frequent; both chronic and acute types.	Very frequent; both chronic and acute types.
Associated symptoms.	Those of infective diseases or intoxications.	Marked pallor of skin, retinitis, bronchitis, etc., inflammations of internal organs.		
Death results from.	Uremia, pulmonary oedema, internal inflammations.	Uremia, or oftener internal inflammations.	Uremia, <i>cerebral hæmorrhage</i> , cardiac failure, internal inflammations.	

Modified from Leube.

AMYLOID DISEASE

Of the kidney, sometimes described as a form of Bright's disease, is not an inflammatory lesion, but a degenerative change. It is, however, frequently associated with interstitial nephritis.

Ætiology.—The disease follows upon prolonged suppuration, especially that due to phthisis or to caries of bone, and upon long-standing syphilis, whether associated with suppuration or not. It occurs, but rarely, in cases of malarial cachexia.

Morbid Anatomy.—The kidney is large (it may be contracted if interstitial nephritis co-exists), pale and smooth on the surface, and firm, the capsule stripping off easily. On section the cortex has a glistening waxy appearance, the medulla being injected. The lardaceous tissues stain mahogany brown with tincture of iodine.

The changes affect first the walls of the vessels (glomeruli and arterioles), spreading thence to the basement membrane of the tubules.

Symptoms vary considerably, according as interstitial nephritis is or is not present. Where it exists, the urinary changes are those already tabulated. Where it is absent, the urine is variable in quantity, pale yellow, *clear*, of normal or low specific gravity, and free from blood. Albumin is sometimes absent or scanty, but usually abundant; sediment is absent, though a few hyaline or waxy casts may be found. *Urea is not diminished*. Dropsy is very considerable, but cardiac hypertrophy, retinitis, and uræmia are all absent, unless there are inflammatory complications. Death occurs from exhaustion, or from the causative disease.

Diagnosis is easy if in the presence of a definite cause, and with evidence of amyloid disease elsewhere (spleen, *liver*, intestine), the above symptoms are to be found. It is difficult when the amyloid change is superadded to an interstitial nephritis. The kidney is never sufficiently enlarged to be accessible to physical examination.

Treatment is that of the causative disease. Cases not too far advanced may recover completely where the cause can be removed (tuberculosis of joints, etc.).

PYELITIS, PYELONEPHRITIS, AND PYONEPHROSIS.

By pyelitis is meant an inflammation limited to the pelvis of the kidney. The inflammation may extend to the substance of the kidney, and there very often leads to suppuration (*pyelonephritis*). Should a large collection of pus form within the pelvis, distending that structure and the calyces, and flattening out the renal substance, the condition is called *pyonephrosis*.

Morbid Anatomy.—If the pelvis alone is inflamed, its mucosa is opaque and thickened, showing punctiform hæmorrhages or superficial ulcers. Pus is present in the pelvic cavity. In pyelonephritis linear areas of suppuration are found extending between the pyramids towards the cortex of the kidney, in which small abscesses are present. In pyonephrosis the wall of the sac is formed by the kidney substance, which is flattened as the result of pressure, and becomes fibrotic from coincident nephritis.

Causes.—Anything which causes severe irritation of the lining of the pelvis of the kidney, such as—

1. Calculi, blood-clots, parasites, tumours.
2. Morbid states of the blood, such as malignant fevers, diabetes, Bright's disease, etc.
3. Certain drugs—cantharides, etc.
4. Extension of inflammation from below (surgical kidney).
5. Pressure upon the ureter, by causing the urine to be dammed back. This urine subsequently undergoing putrefaction may cause intense pyelitis, or pyonephrosis.
6. Injuries to the spinal cord.

Symptoms.—Depend upon the cause, the stage, and the complications. A simple pyelitis gives rise to but few definite

symptoms; a localised pain in the lumbar region may be the chief or only complaint on the part of the patient, or perhaps he seeks advice on account of frequent passage of urine, which, on examination, may be found to contain pelvic epithelia, blood, or pus. When suppuration commences, the symptoms are much more definite: the urine now contains blood, pus cells, and albumin, but remains acid in reaction. There may be occasional attacks of pyrexia, resembling ague. As the kidney *substance* becomes involved, the symptoms of more or less acute nephritis appear. If the suppurative process becomes marked, there are usually rigors, sweating, wasting, and fluctuating temperature. The urine may become ammoniacal, viscid, and "ropy," if the pyelitis has followed upon cystitis. Hæmorrhage is usually slight, but in calculous pyelitis, there may be intermittent profuse hæmaturia.

Sometimes the debris chokes up the ureter, or it may be occluded by a calculus, and the urine which escapes by the free ureter will be clear for a short time, only to become purulent again when the plug moves away. If the pus be pent up, it then causes a cystic swelling of the renal pelvis, that is, "pyonephrosis." It will of course be easily understood that, if such a condition occurred in both kidneys, death would speedily result from uræmia.

The physical signs of pyonephrosis are—

A tumour which (when large) causes distinct bulging in front and behind. Such a tumour

Can be separated from the liver and spleen;

Is always crossed by the colon;

Yields a dull note on percussion; and

Tenderness on pressure.

Prognosis.—Is of course always grave, but recent advances in surgery have rendered the outlook much brighter. If left alone the condition is frequently fatal, but sometimes, even after pyonephrosis has occurred, the pus dries up, the walls of the cyst become coated with patchy layers of phosphatic deposits, and the cavity in time contracts. In other cases the

abscess may burst through the capsule of the kidney, forming a retroperitoneal abscess, which in turn may burrow in various directions, leading to subphrenic abscess, perforation of the diaphragm, etc. Death may also be brought about by extensive lardaceous disease, by cachexia, or by uræmia (typhoid state).

Treatment.—Our treatment should aim to—

1. Remove the cause if possible. In simple pyelitis operative treatment is unnecessary. Rest, light diet, diluent drinks, and urinary antiseptics such as urotropin are sufficient.

2. Relieve the inflamed kidney by rest, cupping, and the administration of sedative salines, such as pot. citras. with hyoscyamus, and the free drinking of liquids.

3. Treat complications as they arise.

Pyonephrosis demands surgical treatment, either by emptying and draining the sac, or in some cases by nephrectomy.

HYDRONEPHROSIS.

A condition due to distension of the pelvis and calyces of the kidney by urine. It may be congenital or acquired.

Ætiology.—Congenital hydronephrosis is usually double, being caused by imperforate urethra. The kidneys may be so dilated as to impede delivery. The acquired form may be single or double. Where it is single it is due to obstruction in the ureter, however caused (stricture, compression by cicatricial bands, impacted calculus, floating kidney, etc.); where it is double, to obstruction lower down, which is usually partial. Thus urethral stricture, enlarged prostate, villous tumours of the bladder, and in the female displacements or tumours of the uterus, may cause it. In such cases death often ensues from uræmia before the renal tumour becomes large enough for recognition during life. In unilateral hydronephrosis the other kidney carries on the renal function. Symptoms dangerous to life do not follow, and the tumour may grow to a very great size.

Morbid Anatomy.—Dilatation of the ureter and pelvis are followed by pressure on the pyramids with flattening and atrophy of the kidney. The sac is divided into loculi by septa formed of the connective tissue surrounding the interpyramidal vessels. There are corresponding bulgings on its surface. The sac contains a pale watery fluid, albuminous, but presenting only traces of urea or other urinary constituents.

Symptoms.—In bilateral cases with incomplete obstruction, frequent passage of very dilute urine, followed later by symptoms of chronic uræmia, and death in the typhoid state. In unilateral cases weight and pain in the loin, and if the tumour be large, nausea, vomiting, or constipation from pressure on the colon. If the obstruction be intermittent, history of sudden passage of a large amount of clear urine, followed by disappearance of the tumour, and gradual re-accumulation. The physical signs are those of renal tumour in general.

Treatment.—Do not interfere if there be no discomfort. Sometimes the sac has been emptied by massage, but operation is usually required. Repeated aspiration may result in cure, but it may be necessary to resort to nephrotomy and drainage, or even to nephrectomy. Make certain first (*a*) that there is another kidney, (*b*) that it is functionally active.

RENAL CALCULUS.

(STONE IN THE KIDNEY.)

Renal calculi are due to the deposition of certain solid constituents of the urine in the substance of the kidney or in the renal pelvis. They may be of any size, forming granules of "sand" or "gravel," or large concretions. They may be solitary or very numerous.

Characters.—Calculi of appreciable size are made up of concentric layers arranged around a nucleus often of quite different composition. The nucleus may consist of crystals of uric acid or oxalate of lime, or of colloid bodies such as

mucin, epithelia, or tube casts. The laminæ are composed of uric acid (the commonest form), oxalate of lime, alternate layers of uric acid and oxalate, or sometimes phosphate of lime and triple phosphates deposited upon a nucleus of one of the other crystals. This occurs in the presence of ammoniacal decomposition of the urine in the pelvis, as sometimes in pyonephrosis. Other forms of calculi (cystine, xanthine, etc.) are very rare.

Uric acid calculi are of a reddish colour, hard, smooth, and, if they are multiple, faceted on the opposing surfaces. Oxalate calculi are very hard, greyish, smooth, and rounded while small, but when they become larger presenting a rough tuberculated surface ("mulberry calculi"). Phosphatic calculi may grow to a great size by continual deposition of phosphates from the alkaline urine. They tend to assume the shape of the pelvis and calyces, and form "dendritic" masses.

Ætiology.—The deposition of uric acid is favoured by increased acidity of the urine, and diminution of the salts, especially phosphates, which tend to hold it in solution. Oxalates tend to be deposited when there is excess of them in the urine. But neither uric acid nor oxalate crystals agglomerate into calculi without some additional factor, which is supplied by the presence in the pelvis of colloid material such as mucus, albumin, or blood. These may be present as the result of slight catarrhal pyelitis or congestion of the pelvis. That such local causes are at work is shown by the frequency with which calculus is unilateral.

Morbid Anatomy.—A calculus generally sets up some degree of pyelitis, which may be suppurative, and lead to pyonephrosis. If the ureter be obstructed, the kidney may atrophy, or hydronephrosis may follow. Malignant tumours have been attributed to the continued irritation of the calculus. If both ureters be obstructed, anuria and the symptoms of latent uræmia will follow.

Symptoms may be latent throughout life. The most characteristic is renal colic, but between the attacks there may

be (1) pain in the lumbar region, aching or dragging, and made worse by movement; (2) some degree of tenderness on deep pressure in the lumbar region; (3) hæmaturia, recurring at intervals, often without other symptoms, aggravated or induced by movements especially of a jolting nature (riding, jumping, etc.), and relieved or removed by rest; (4) marked acidity of the urine, which may contain uric acid or oxalate crystals, and, if there be pyelitis, pus.

Renal colic results from the passage or attempted passage of a calculus down the ureter to the bladder. It is characterised by excruciating pain beginning in the loin and radiating over the anterior aspect of the abdomen on the affected side, and downwards to the testicle and the inner side of the thigh. The skin is hyperæsthetic over the affected area. The patient is faint, and sweats profusely. The pulse is small and rapid; there are nausea and vomiting; and pregnant women may abort. The paroxysm may last a few hours or a few days. It ends suddenly if the stone slips into the bladder; more gradually if it becomes impacted. During the attack urine is passed frequently and in small quantity. It is usually scanty, high-coloured, and blood-stained. It may be suppressed.

Where the opposite ureter is already obstructed, death may ensue from *obstructive suppression*. There is anuria, without marked symptoms beyond lassitude and insomnia for about a week. Then muscular twitchings appear, the pupils become contracted, and there is gradually increasing dyspnœa, which leads to death within a day or two. The mind remains clear till just before death.

Treatment.—Uric acid calculi are soluble in alkaline solutions. The urine should therefore be rendered alkaline by salts of potassium such as the citrate or acetate (5j - 3ij every three hours), and the treatment should be kept up for three months or more. The diet should be light, as little nitrogenous as possible, and stimulants should be avoided. Oxalate calculi are not affected by this treatment. In them, and in uric acid calculi where the solvent treatment has failed, operation (nephrotomy or nephrectomy) may be required.

The treatment of renal colic is to be directed to the relief of pain, by hot fomentations, hot baths, morphia, and, if necessary, chloroform.

RENAL TUBERCULOSIS

May occur in the form of miliary tubercles in the course of a general infection, or may be primary. The disease may begin in the kidney and spread downwards, or may extend to the kidney from the lower urinary tract, in which case both kidneys are usually affected.

Morbid Anatomy.—The primary form of the disease begins by the formation of tubercles in the medulla, which coalesce and lead to caseation and softening. Cavities are thus formed, separated from each other by inter-pyramidal septa of fibrous tissue. The whole renal substance may be ultimately destroyed. The pus is discharged in the urine, causing tuberculous pyelitis, extension of the process to the ureter, and consequently partial or complete occlusion of that structure, leading to pyonephrosis.

Symptoms are chiefly those of pyelitis, and consist of dull aching pain in the loin, frequent micturition, constant presence of pus in an acid urine, proportionate albuminuria, and often hæmaturia, much less readily influenced by rest than that of calculus. Tubercle bacilli *may* be found in the urine. There are the usual general symptoms of wasting, fever, and sweating; later pulmonary or peritoneal tuberculosis may appear. If pyonephrosis is present there may be evidence of renal tumour; tuberculosis *per se* does not materially increase the size of the kidney.

Treatment, medicinal and hygienic, is that for tuberculosis in general, with the addition of such urinary antiseptics as salol, urotropine, or benzoate of soda. Nephrectomy can only be thought of where the disease is unilateral and the bladder is unaffected; nephrotomy and drainage may sometimes be of use.

RENAL TUMOURS

Are almost invariably malignant. Sarcomata are congenital tumours of complex structure, often growing to an enormous size, and running a brief course to death. The tumours of adult life are either epitheliomata originating in the pelvis, or adeno-carcinomata originating in the cortex. The former cause uniform enlargement of the kidney, the latter nodular out-growths. There may be direct extension through the capsule of the kidney to the retroperitoneal tissue; or extension may take place by the veins (the renal vein or inferior cava may be blocked by the growth) to the lungs, or by the lymphatics to the liver and lumbar glands.

Symptoms are (1) lumbar *pain*, dull and aching, but sometimes like that of renal colic, where blood-clot is present in the ureter; (2) *tenderness* on pressure; (3) recurrent *haematuria*, often profuse; (4) sometimes *varicocele*, from pressure on the spermatic vein; (5) evidence of tumour; (6) the general symptoms of malignant disease.

Characters of a Renal Tumour.—(1) The tumour fills up the loin, and bulges forwards. Its shape is rounded, and its edges blunt. Its direction is vertical. (2) The fingers can be slipped beneath the ribs above it (distinction from liver or spleen), and beneath the iliac crest below (distinction from ovarian tumour). (3) It is only slightly movable on respiration. (4) The colon may be traced in front of it by palpation or percussion (see that the bowel is emptied before examining). (5) The dulness is continued posteriorly to the middle line (distinction from spleen). (6) It may cause *pressure symptoms*—(a) varicocele, (b) ascites from pressure on the portal vein, (c) enlargement of superficial veins from pressure on the vena cava, (d) intestinal obstruction.

Treatment, as far as it is not surgical, is purely palliative.

MOVABLE OR FLOATING KIDNEY.

The kidney may be unduly mobile from laxity of the sub-peritoneal fascia. The peritoneum may nearly surround it, forming a false mesonephron. This condition may be part of a general enteroptosis, or may be the result of laxity and pendulousness of the abdominal wall. Tight lacing is said to cause it. It is more frequent in the female than the male.

Symptoms may be entirely absent. There is often dragging pain in the loin, worse on exertion. Paroxysmal crises of pain like that of renal colic, attended by rigor, vomiting, and collapse, may occur (Dietl's crises). Neurasthenia and dyspepsia are frequent. Intermittent hydronephrosis may occur from temporary kinking of the ureter. A freely movable tumour of the size and shape of the kidney is to be felt. It may occupy almost any position in the abdominal cavity. Squeezing it causes a feeling of nausea. It can be pushed back into the loin by palpation.

Treatment.—If a displaced kidney causes no symptoms, and is found by accident in the course of an abdominal examination, the patient should not be told of its existence. No treatment may be required, but in other cases a carefully adjusted pad and bandage may retain the kidney in position. Sometimes it is necessary to stitch it to the posterior wall.

EXAMINATION OF THE URINE.

The examination of the urine in disease is of the utmost importance. Where quantitative analysis is required, a specimen *of the urine passed in the twenty-four hours should be used*, as obviously the urine is richer in certain constituents at certain parts of the day. Observe—

1. *Quantity.*—Normal, 45 to 52 ounces (1300 cc. to 1500 cc.).

2. *Specific Gravity.*—Normal, 1015 to 1025. If above

1025, test for sugar. If necessary, estimate the total amount of solids from the sp. gr. and quantity.

3. *Odour*.—The normal odour is peculiar and characteristic. It becomes ammoniacal and putrid when the urine decomposes. It smells like honey when sugar is present, and like sweet violets after the administration of turpentine.

4. *Reaction* is normally acid, except after a meal containing much vegetable food. Decomposed urine is alkaline.

5. *Colour*.—Normal, pale straw to dark amber.

(1) If red, or reddish-brown, or smoky, suspect presence of blood, and search for blood corpuscles; test with guaiacum and ozonic ether, and if necessary, examine with the spectroscope.

(2) If greenish or yellowish-brown, suspect presence of bile pigment; observe colour of froth; test for bile pigment and bile acids.

(3) If pale, quantity large, and sp. gr. over 1025, test for sugar.

6. *Deposit*.—Allow the urine to settle in a cylindrical or conical glass, and a cloud of mucus invariably forms. It is always light, and moves easily with the fluid.

A deposit of urates is the next most common. It is usually of a brick-dust colour, and moves easily when the vessel is inclined.

A deposit of earthy phosphates has usually a white or dirty-white appearance, and is somewhat heavy.

If the deposit be slimy, it probably contains much mucin or pus.

If the urine is milky on passing, it is usually due to deposit of the earthy phosphates. It is common after a full meal, especially when vegetables have been freely partaken of; the urine in such a case is usually neutral or alkaline. Do not mistake this deposit for pus, nor confound *earthy* phosphates with triple phosphates.

	URATES.	EARTHY PHOSPHATES.
<i>Colour</i> . . .	Usually brick-red.	White or milky.
<i>Heat</i> . . .	Usually clears up.	Unaffected.
<i>Alkalies</i> . . .	Dissolve.	Do not dissolve.
<i>Acids</i> . . .	Insoluble.	Soluble.

Albumin.—The tests for albumins depend on their coagulability by heat and precipitation by nitric acid, picric acid, etc. The most reliable tests are—

1. *Heller's Test.*—Put a little nitric acid in a test-tube, pour the albuminous urine cautiously upon it, and an opalescent ring of coagulated albumin appears above the acid. If the albumin is abundant it appears quickly; if there is only a trace of albumin its appearance may be delayed for two or three minutes.

But nitric acid in the cold also precipitates *albumose*; this is distinguished from albumin by the precipitate dissolving when heated and returning when cooled.

In using Heller's test remember that the addition of nitric acid to undiluted urine may cause a precipitate of *uric acid*; this, however, is always very scanty, and the microscope shows crystals. Further, if the urine be very concentrated the addition of nitric acid may cause a precipitate of nitrate of urea, but its appearance is very different from that of the flocculent precipitate of coagulated albumin. Nitrate of urea is not thrown down when the urine is diluted before adding the acid.

2. Boil the urine in a test-tube. A flaky precipitate falls, which may be albumin, or if the urine is only faintly acid, phosphates. Phosphates are re-dissolved on acidulating with acetic acid, albumin is not.

Picric acid and salicyl-sulphonic acid are also used as tests.

3. *The quantitative test* may be roughly performed by Esbach's Albuminimeter; but since picric acid is the reagent

used, it should be remembered that *all the proteids* in the urine, other than albumin, are thereby precipitated.

METHOD.—*Esbach's Albuminimeter* is a graduated tube for roughly estimating the percentage of albumin in urine. It cannot be successfully used if the percentage is large. It succeeds best when the urine is diluted until its sp. gr. is not above 1010. If necessary, therefore, an equal volume or two volumes of water may require to be added to the urine, and this must be taken into account in making the calculation.

1. Fill tube with urine up to mark *U*.
2. Add picric and citric acid solution to mark *R*.
3. Mix thoroughly, and set aside for twenty-four hours.
4. Read height of coagulum on scale. The numbers on the scale indicate grammes of albumin per litre of urine. Result only approximative, because the bulk of the coagulum depends much upon its density.

The most accurate method of determining the exact quantity of albumin is Brandberg's modification of Sir W. Roberts' method; the process is a tedious one, and requires much practice to be accurate. (For details of this test see von Jaksch's *Handbook*.)

Globulins in the urine are precipitated by a saturated solution of magnesium sulphate.

Peptones are tested by the Biuret test—*i.e.*, giving a pink or violet reaction with KOH and a *trace* of sulphate of copper. Peptones are not coagulated by heat.

Sugar or Dextrose in the Urine.

The various tests used are those of Moore, Fehling, Trommer, the phenyl-hydrazin test, the fermentation test, and the test by means of the polarising saccharometer.

Fehling's solution is an alkaline solution of potassio-tartrate of copper, and is made of such a standard strength that 1 cc. of *recently* prepared Fehling's solution is reduced by 5 milligrammes of grape sugar. It may be used to demonstrate the

presence of a reducing sugar, or for the quantitative estimation; but in the latter case it is better to use the Pavy-Fehling solution, which contains ammonia in addition to the other salts. The ammonia prevents the precipitation of cuprous oxide, and thus the complete reduction is indicated at once by the disappearance of the blue colour of the reagent.

METHOD.—Having removed albumin (if present) from a sample of the urine passed in twenty-four hours—

1. Dilute the urine to 1 in 20 (5 cc. urine—95 cc. water).
2. Place the urine in a burette.
3. Measure 10 cc. Pavy-Fehling solution, and place in porcelain basin or small flask.
4. Heat to boiling, and while boiling drop in urine until blue colour just disappears. If the urine be dropped in too slowly, the ammonia may be driven off by the prolonged heat, and a precipitate of cuprous oxide will fall, confusing the reaction.
5. Calculate the total amount of sugar in the urine passed in twenty-four hours.

For details of the other tests consult a work on Examination of the Urine.

It must not be forgotten that traces of sugar may exist in healthy urine.

Acetone.—Add to the urine a few drops of a fresh saturated solution of sodium nitro-prusside and then a few drops of NaHO. A dark red colour appears. Acidulate with acetic acid, and the red changes to crimson. Acetone does not strike a Bordeaux red with ferric chloride, thus differing from diacetic acid. (See Diabetes.)

Bile in the Urine.—

Bile pigments give from a deep orange or greenish to almost a porter-like hue to the urine; it froths easily, *the froth being tinged with the pigment*.

TESTS.—1. Add impure nitric acid (nitric-nitrous) cautiously to the urine in a test-tube or on a porcelain slab; at the

junction of urine and acid a display of colours occurs, due to oxidation of the pigments. The characteristic colour is green.

2. Pour on to the surface of the urine in a test-tube a dilute solution of tincture of iodine (1 in 10). A green ring forms at the point of contact.

Bile Acids.—Cannot be detected in bilious urine without evaporating, or unless the amount of bile is excessive.

METHOD.—Add a solution of cane sugar to the urine in a test-tube, shake, let a drop of H_2SO_4 trickle down the tube; at the junction of the acid and froth a cherry rose-red colour will develop. The test is very fallacious.

Blood in the Urine.—(See page 338.)

Urea.—The quantitative test is of the utmost importance; for by comparing the amount of nitrogenous material taken in, with the amount of urea passed, we are enabled to gauge the state of metabolism in the body. Urea is increased during active metabolism as in fevers, etc., and is of course decreased when the metabolic functions are in abeyance. It must not be forgotten, however, that the formation of urea may be for a time excessive, and yet the excess may not appear in the urine through inability of the kidneys to excrete it.

Volumetric Estimation of Urea by the Hypobromite process depends on the fact that the solution of sodium hypobromite decomposes urea into CO_2 , N, and H_2O . The CO_2 combines with the free soda, and nitrogen, therefore, alone passes into the burette. One gramme of urea by this method yields 371 cc. of nitrogen, therefore, 37.1 nitrogen equals 1 decigramme of urea. If we measure, therefore, the amount of N given off from a known quantity of urine, we can calculate the total amount of urea passed daily.

APPARATUS. Burette inverted in tall jar of water. Burette connected by elastic tube with a small glass flask in which urine is to be decomposed. A jar with water to cool urine and gas during decomposition. A glass tube to contain a measured quantity of urine.

PROCESS—

1. Place about 15 cc. hypobromite solution in flask.
2. Place 5 cc. mixed urine of twenty-four hours in small tube, and introduce it into flask without tilting.
3. Elevate burette in jar till water inside it is at the 50 cc. line. Cork the flask, and read position of water in burette again.
4. Tilt flask slowly and mix the urine with the hypobromite. Jar should be left to cool for ten minutes.
5. Elevate burette till water inside is on the same level with that outside, and read off gas, then calculate amount of urea present in 5 cc. urine, and then the amount in the total urine in twenty-four hours.

For perfect accuracy, the volume of the gas should be corrected for temperature and barometric pressure.

The recently introduced ureameter of Doremus, manufactured by Southall, is a much more convenient instrument than that described, and is quite sufficiently accurate for clinical purposes.

It would be useless to calculate the amount of urea passed per day if we did not know how much *should* be passed. All things being equal—*i.e.*, an average diet, average labour, and average health—the excretion should at least reach three grains for every pound weight. In other words, a man weighing 150 lbs. should excrete at the lowest 450 grains of urea; he might with advantage excrete up to 500 grains, but it should not fall less than 450 grains per diem. The amount of urea is always diminished in vegetarians.

Uric Acid appears in the urine to the extent of 7 to 12 grains in combination with soda, etc., as urates. It is much increased in fevers, deficient action of the liver, gout, etc. In order to test for its presence we first displace it from its base by adding an acid, HCl, or acetic acid, and letting the urine stand awhile. When uric acid is deposited it forms a cayenne pepper or red sand-like deposit.

Tests for Uric Acid. —1. *Garrod's Thread test.* Concentrate

fluid. Place 5 cc. in a watch-glass. Add ten drops glacial acetic acid. Place thread in fluid, and leave in cool place for twenty-four hours to allow crystals to form.

2. *Murexide test.* Place five drops urine in porcelain capsule. Add one drop nitric acid. Evaporate *gently* nearly to dryness. Add small drop of ammonia = purple colour, due to murexide or purpurate of ammonia.

3. *The Volumetric test.* The acid is estimated by Haycraft's method. Uric acid is made to combine with silver, forming a gelatinous precipitate; this is separated by filtration and made into a solution with nitric acid. The amount of silver is then tested by a colour test, and the amount of uric acid calculated by the amount of silver found.

Chlorides.—The amount of *chlorine* excreted daily amounts to about 100 grains, chiefly in combination with soda.

Test.—Silver nitrate preceded by acidulation with nitric acid.

The chlorides are markedly diminished in all cases of croupous or fibrinous inflammations; this is particularly the case in croupous pneumonia.

Quantitative Estimation.—

Silver Method.—*The solutions required are—*

1. Solution of *silver nitrate*, containing 29·075 grms. of the fused salt in 1000 cc. of distilled water; of this solution 1 cc. = 0·01 grms. of sodium chloride.
2. Saturated solution of *potassium chromate* (neutral).

METHOD.—Take 10 cc. of the urine, and dilute with 100 cc. of distilled water. Add to it a few drops of solution (2). To this mixture in a beaker allow the standard solution (1) to drop in from a burette. A precipitate of silver chloride will occur as long as any chlorine is uncombined. When the whole of the chlorine is satisfied, a reddish or pink (since there is much white precipitate present) precipitate of silver chromate appears. This indicates the time to stop the addition of the silver nitrate, and the amount of the solution which has been used is read off. This will indicate the amount of silver nitrate necessary to

convert all the chlorine present in 10 cc. into silver chloride. It is known that 1 cc. of the solution = 0.01 gm. of salt, and from this the total amount of chloride present can be estimated. A correction should be made by subtracting 1 cc. of the silver solution used, as the urine contains certain other substances more easily precipitated than the chromate. (Power and Harris's *Handbook*.)

Phosphates.—The tests for phosphates generally have already been considered. The test for orthophosphoric acid is precipitation by uranium acetate, the precipitate being insoluble in acetic acid.

Pus.—

1. Examine under microscope for pus cells.
2. KOH added to the urine causes it to become stringy.

For the various casts and calculi found in the urine, see a work on Examination of the Urine. (Beale's plates are excellent.)

DISEASES OF THE NERVOUS SYSTEM.

ANATOMY AND PHYSIOLOGY OF THE SPINAL CORD.

THE reader is strongly advised to digest the following brief account of the anatomy and physiology of the motor and sensory tracts, before commencing the study of the various diseases of the nervous system. I shall not attempt anything approaching an anatomical description, but content myself with the briefest description of those facts *which form the foundation of a clear understanding of clinical phenomena attendant upon pathological changes.*

The Spinal Cord consists of a tubular prolongation of the brain enclosed in three membranes—

1. Dura mater, externally.
2. Arachnoid, in the middle.
3. Pia mater.

Between the arachnoid and pia mater is the sub-arachnoidal space, filled with cerebro-spinal fluid. It should be noted that this space is continuous with the ventricles of the brain through the foramen of Majendie. The cord substance shows *white* matter externally, and grey matter internally. Like *all nervous tissue* the white and the grey matter have as a ground work *neuroglia*, or nerve glue.

Neuroglia shows under the microscope—

1. Large and small cells ("Deiter's cells").
2. Granules.

3. Fine and very numerous fibres, crossing in all directions.
4. Interstitial cement substance.

Note that "sclerosis" means a great increase in neuroglia at the expense of the special nerve elements.

The *grey matter* differs from the white in—

1. Colour, and being more vascular.
2. Having special groups of nerve-cells arranged—

Anteriorly	} termed {	Anterior vesicular column.
Laterally		Lateral vesicular column, or inter-medio-lateral tract.
Posteriorly		<i>Posterior column or column of Clarke.</i>

The *anterior cells* are multipolar and give origin to motor nerve-roots; *they also act as trophic centres for motor fibres in the nerve trunk.*

The posterior cells (or column of Clarke) are very important through being implicated in the passage of sensory fibres, as will be seen farther on.

The *white matter*, though apparently homogeneous, is really mapped out into columns. Each column is specially concerned in transference of certain impulses up to, or from, the brain. The tracts through which these impulses are conducted are known as sensory, afferent, or ascending tracts; and motor, efferent, or descending tracts. Patient investigation has shown that there are at least two purely motor or *descending* paths, and four purely sensory or *ascending* paths.

The nerve-cells with their processes (axon and dendrons or dendrites) constitute the primary conducting elements of the nervous system, and are known as *neurons*. The places of linkage of the neurons are called *synapses*. There is often an intercellular gap between adjacent neurons. In the motor tracts there are two sections—(1) An upper or central neuron, comprising the cortical motor cell, its dendrons, and the axon or chief process, which passes downwards through the cord to terminate in an arborisation round a motor cell of the anterior grey cornu; (2) a peripheral neuron, comprising the motor cell of the anterior cornu, its dendrons and its axon,

passing downwards to the muscle and ending on the muscular fibres. The sensory path is by the ganglia of the posterior roots.

THE TRACTS.

(See Diagram, p. 375).

Motor, Efferent, or Descending Tracts—

1. Direct Pyramidal situated anteriorly.
2. Crossed Pyramidal situated laterally.

Sensory, Afferent, or Ascending Tracts—

- | | |
|--|----------------|
| 1. Comma Tract of Gowers. | } Laterally. |
| 2. Direct Cerebellar. | |
| 3. Postero-External, or column of Burdach. | } Posteriorly. |
| 4. Postero-Internal, or column of Goll. | |

The lateral limiting layer and the antero-lateral ground bundle contain both ascending and descending fibres.

CEREBRO-SPINAL NERVES.

An ordinary mixed nerve is made up of—

1. Anterior or motor root fibres.
2. Posterior or sensory root fibres.
3. Sympathetic fibres.

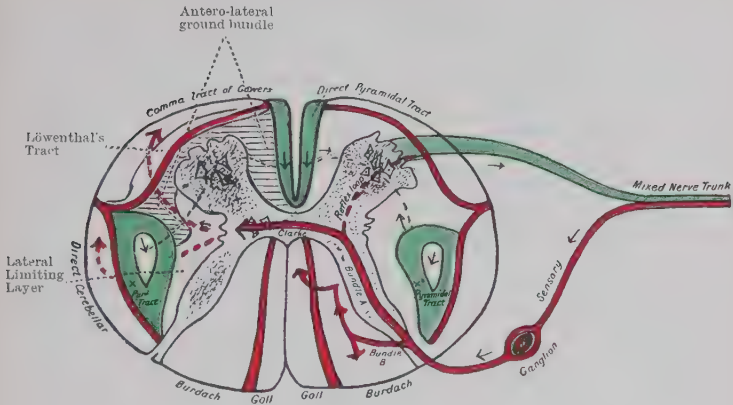
The *branches* of such a nerve may contain only (1) motor fibres, or (2) sensory, or (3) principally sympathetic.

COURSE OF MOTOR FIBRES.

As motor fibres proceed *from* the brain, we shall describe them from *above downwards*.

Starting from the cortex (central neuron), principally around the *fissure of Rolando*, they proceed through—

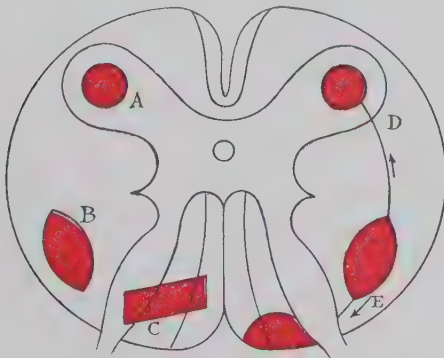
1. Corona radiata.
2. Anterior two-thirds of *posterior* limb of internal capsule.
3. Crusta of Crus.
4. Pons Varolii.
5. *Medulla*, where the bulk of the fibres cross (decussation of pyramids) to the opposite side, and pass down the cord as



A DIAGRAM, to show the course of the motor fibres (green) and the sensory fibres (red) in the cord and nerve roots.

Note the motor fibres pass from the direct and crossed pyramidal tracts to the anterior horn of grey matter, and thence to the anterior or motor nerve root.

The sensory fibres pass in, in two bundles, A and B. Observe bundle A crosses to the opposite side, a few fibres, however, going to the anterior horn to form the reflex loop; bundle B passes into Burdach first, then at a higher level into Goll's tract (so that some sensory fibres cross to the other side at once, others do not).



DIAGRAMMATIC DRAWING to show the fibres involved by lesions at A, B, C, D, and E.

A, Progressive Muscular Atrophy.

C, Locomotor Ataxia.

B, Spastic Paralysis.

D, Amyotrophic Lateral Sclerosis.

E, Spastic Ataxia.

the *crossed pyramidal tract*, thence to the multipolar cells of the anterior horn of grey matter, and finally out by the *anterior motor roots* (peripheral neuron). A few fibres pass down from the medulla without decussation, and so form the direct pyramidal tract.

NOTE here the trophic centre for the *motor tracts* is situated in the cortex, happily termed the first *trophic realm* by Wyllie. The trophic centres for the *motor nerves* are, as before stated, the multipolar cells in the anterior horn of grey matter, named by Wyllie the second *trophic realm*, or putting it briefly, a lesion of the motor cortex is followed by degeneration of *motor tracts*. A lesion of the anterior grey horn is followed by degeneration of motor *nerves*, plus other changes.

SENSORY FIBRES.

Formerly they were supposed to enter the cord and immediately decussate over to the opposite side. Recent researches show that while there is still much to be discovered with regard to sensory tracts, the more important paths of the sensory fibres in the cord are clearly defined. The following statements form a good working basis.

Sensory fibres enter the grey matter in two bundles—*i.e.*, Bundle *A* and Bundle *B* (see diagram, p. 375).

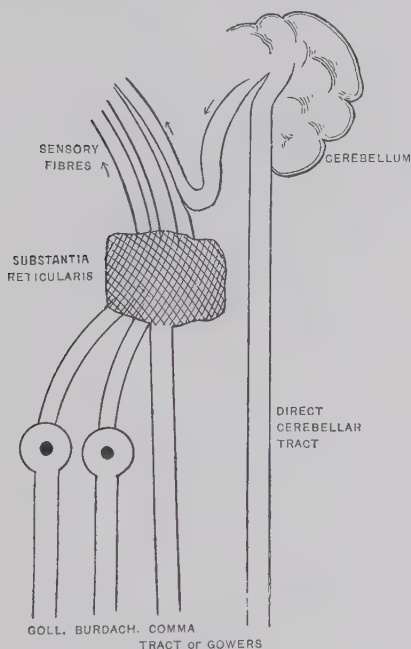
Bundle B passes first into column of Burdach, descends and ascends a little, then enters the column of Goll, passes straight up to the medulla, and there decussates to the opposite side.

Bundle A passes into the grey matter. A few fibres go to the anterior horn to form, with the motor cells, a *complete reflex loop*. The bulk of the fibres, however, cross to the column of Clarke, and thence to direct cerebellar and comma tract of Gowers, and to the antero-lateral ground bundle.

Thus in the cord we have four distinct sensory railroads—*viz.*, the columns of Burdach and Goll, the tract of Gowers, and the direct cerebellar tract—as high as the medulla oblongata. It is not known definitely how they ascend from the medulla. For clinical purposes, however, we may say that sensory fibres

are scattered throughout the brain substance, though possibly the larger number proceed as follows—

1. The direct cerebellar tract goes direct to the cerebellum.
2. The tract of Gowers to the substantia reticularis.



DIAGRAMMATIC SCHEME, by the writer, to show a possible rearrangement of the Sensory Tracts in the Medulla before passing up to the Cortex as sensory fibres. (See pages 376 and 377.)

- 3 & 4. The tracts of Goll and Burdach end as clavi in the medulla.

From each clavus fibres pass to the substantia reticularis. Thus the tracts of Goll, Burdach, and Gower are amalgamated at a common junction. From this junction they pass upwards (behind the motor fibres) as *sensory fibres*, strengthened by

co-ordinating fibres from the cerebellum, their course being through—

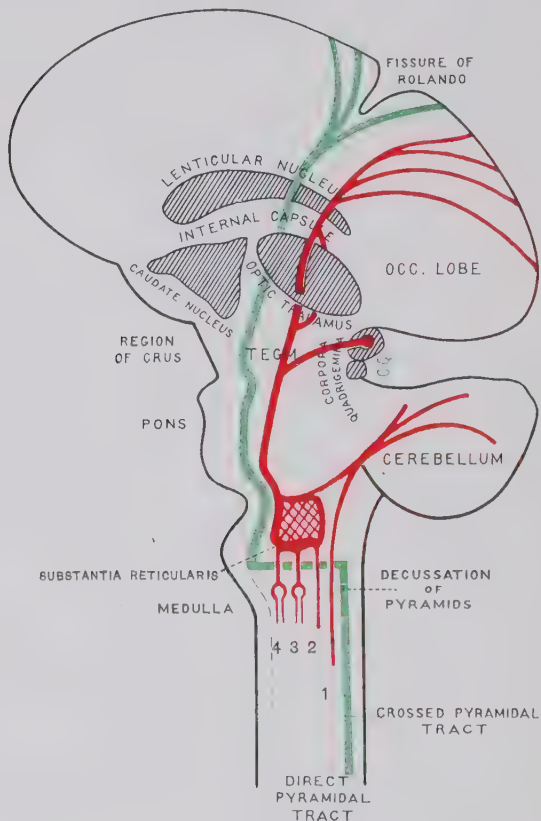


DIAGRAM to show the course of the principal motor (*green*) and sensory (*red*) fibres between the medulla and cortex.

1. Medulla.
2. Pons.
3. Tegmentum of crus.
4. Posterior third, *posterior* limb of internal capsule.

5. Corona radiata.
6. Cortex, especially occipital lobe.

(A glance at the diagram will make this intelligible.) Some of the fibres stop, *en route*, at the corpora quadrigemina and optic thalamus.

The internal capsule, through which the motor and sensory fibres pass, is the band of white matter between the basal ganglia, its boundaries being—

Externally = Lenticular nucleus.
Internally = { Caudate nucleus (anteriorly).
 { Optic thalamus (posteriorly).

In the middle of the internal capsule is a “bend” termed the “genu” or knee; the portion in front of the knee is called the anterior limb; the portion behind, the posterior limb. The former contains commissural fibres of various kinds, the genu contains descending fibres passing to the cranial nuclei of the opposite side, whilst the *posterior* limb contains, as we have already seen, the *sensory and motor fibres* from the spinal cord.

A lesion of the internal capsule involving the motor fibres must therefore cause motor paralysis of the side of the face and of the limbs on the opposite side of the body.

Sensation is not usually so much affected, as it will be readily seen that a lesion, extensive enough to cause both *sensory and motor* paralysis, would probably be fatal.

The external capsule, as its name implies, is outside the internal capsule, and is that portion of the cerebrum lying between the—

Clastrum, externally;
Lenticular nucleus, internally.

It is in close proximity to the Island of Reil.

GENERAL SYMPTOMATOLOGY.

REFLEXES.

The student is frequently puzzled as to when reflexes should be lost, exaggerated, or impaired, but if he bears the following facts in mind there should be no difficulty.

A reflex act requires a continuity between an *afferent* nerve, and an *efferent* nerve. The parts usually involved are—

1. A receiving surface (as skin).
2. A sensory nerve (afferent fibre).
3. A receiving station (cells in cord).
4. A motor nerve (efferent fibre).

These together constitute a *reflex loop*. A break in *any* part of this loop must be attended with loss of reflex. What is the effect of the brain on reflexes? The *brain* exercises an inhibitive action; therefore, if the cord be cut off from its inhibitive influence, reflexes must be exaggerated.

Imagine the cord to be built up of segments, each segment giving off a pair of spinal nerves; then take a transverse lesion of the cord, what state of reflexes would we get?

1. At point of lesion—reflexes are lost (as the reflex loop is destroyed).

2. *Below* lesion—reflexes are exaggerated (as inhibitive action of brain is cut off).

3. *Above* the lesion—reflexes are normal.

Note, however, at the upper border of the lesion, the dead part would act as an irritant to the healthy portion of the cord, and consequently cause irritation of any sensory nerves coming off that area, causing a band of hyperæsthesia.

This painful zone, taken in conjunction with the condition of reflexes, often enables an accurate localisation of the lesion to be made.

The hyperæsthetic zone also explains the girdle pain present in many lesions of the cord.

DEEP OR TENDON REFLEXES.

These are not true reflexes, but muscular contractions produced by direct excitation of the muscle or its tendon. They are dependent upon the tonus of the muscle, which in its turn depends upon the integrity of the reflex arc. Thus disease affecting the afferent or efferent parts of the loop, or the receiving station in the cord, abolishes them; and they are exaggerated when the cerebral inhibitory action is interfered with.

The *knee-jerk* is present in health. It is obtained by tapping the patellar tendon when the quadriceps femoris is slightly stretched by the patient crossing one leg over the other.

Knee-clonus is obtained by pulling down the patella as the patient lies upon his back, and delivering a tap upon its upper edge while the tendon is stretched. It is not present in health.

Ankle-clonus is obtained by supporting the calf of the patient's leg upon one hand, and thrusting the foot upwards with the other hand placed upon the ball of the toes. It is also absent in health. A triceps tendon reflex may sometimes be obtained when the deep reflexes are exaggerated.

DEGENERATIONS.

When nerve fibres are cut off from their trophic centres, they undergo disintegration, known as secondary degeneration, —viz., in the case of sensory fibres = ascending degeneration, and in the case of motor fibres = descending degeneration.

In the case of sensory fibres, the trophic cells are situated peripherally, and the direction of the axon is centripetal. Hence in the cord sensory degeneration starts from below and passes upwards towards the cerebrum. In the motor tracts the reverse is the case, as the trophic cells have a central situation. Degeneration therefore occurs from the cerebrum downwards through the cord, or from the anterior cornu along peripheral nerve fibres.

Such degenerative changes in the motor tracts express themselves in an alteration of the electrical reactions of the muscles governed by the affected nervous area, and also by alterations in the reactions of the implicated nerves. These are known as the reaction of degeneration, or briefly, R. D. Tested by electricity, the changes briefly are (on applying the electrodes to the *muscle*)—

1. Increased excitability to galvanic and faradic currents; this quickly passes off, and we get
2. *Decreased* to faradic, but *increased* to galvanic; next
3. Decreased to *both* galvanic and faradic.
4. Lost to both, in cases where the nervous disease is permanent. In other cases the response to faradism and to galvanism gradually returns.

These peculiar changes are accompanied by “polar” alterations.

In health we get in response to a minimum current—

1. K.C.C. = kathodal closing contraction is strongest; next,
 2. A.O.C. = anodal opening contraction
 3. A.C.C. = anodal closure contraction
 4. K.O.C. = kathodal opening contraction, the weakest.
- } equal.

But in disease—

A.O.C. or anodal opening contraction may be stronger than the K.C.C. (kathodal closing contraction)—*i.e.*, a reversed condition to that seen in health. The main point, however, to remember is, “that a *muscle* cut off from its nerve, or supplied by a *diseased* nerve, undergoes—

1. Increased excitability.
2. Decreased excitability, and finally fails to respond to any electrical stimulus.”

Pathology of R. D.—The nerve structure shows—

1. Breaking up of white substance of Schwann.
2. Nuclei, swollen and granular.
3. Axis cylinders broken, and nerve substance transformed into fibrous cords.

The Muscle structure shows—

1. Muscular fibres atrophied.
2. Disappearance of transverse striæ.
3. Granules.
4. Great increase of fibrous tissue.

SPASM AND RIGIDITY.

When a motor nerve is irritated, the muscles supplied by that nerve contract. If the irritation be kept up, the contractions are more or less constant, and cause "spasm." Spasmodic muscular contractions may be of two kinds, *tonic*, when the contractions are continuous, and *clonic*, when they are intermittent. Spastic contractions of the muscles of the lower limbs, especially when the patient is falling asleep, and cerebral control is thereby relaxed, are frequent in early degenerative lesions of the motor tract. Rigidity is also produced by irritation of motor nerves. It is of two kinds. When the motor tracts are injured, let us say by a severe hæmorrhage in the brain, there is not only paralysis, but also rigidity of the limbs quickly supervening, *due to irritation of the motor fibres*; this is termed "early rigidity," and soon passes off.

Suppose the patient recovers from the fit of apoplexy, with, however, a *resulting hemiplegia*, it will be noted that later a second and permanent rigidity takes place. This is also due to irritation, but of a *destroying nature—i.e.*, secondary descending degeneration.

Rigidity of a similar nature may also occur independently of cerebral disease, when the motor tracts in the cord are affected by sclerosis (lateral sclerosis, disseminated sclerosis).

Irritation, then, gives rise to increased nerve action; and *compression*, on the other hand, causes paralysis.

CO-ORDINATION AND INCO-ORDINATION.

By *co-ordination* is meant the harmonious action of muscles involved in the carrying out of complicated movements.

Inco-ordination means failure of this harmony.

The cerebellum is one of the *chief* centres for co-ordinated movements, though there are subsidiary centres. The centres seem to act through afferent impressions derived from—

1. The sense of touch (columns of Goll and Burdach).
2. Sight (optic nerves).
3. Auditory organs (semicircular canals).

Lesions interfering with these afferent fibres are attended with more or less inco-ordination. Inco-ordination may, therefore, be due to—

1. Disease of the *centres* rendering them powerless to emit the necessary influence.
2. Disease cutting off the means (afferent fibres) by which the centres are stimulated.

SCLEROSIS.

Sclerosis is a convenient term used to express the pathological changes which take place in *chronic degenerative lesions* of the nervous system. We shall draw a picture of a typical "sclerosis," so as to avoid a constant repetition of words, then, in describing each particular disease, merely point out the *sites* of such lesions.

Sclerosis.—*Grey Matter shows—*

1. Increased fibrous tissue (neuroglia).
2. Atrophy of *proper nerve-cells*.
3. *Blood-vessels thickened*, some obliterated.
4. Extravasation of pigment.
5. Small patches of fatty degeneration.

White Matter shows—

1. Discoloration.
2. Absence of myelin.
3. Great increase of fibrous tissue.
4. Granular debris.

Roots, when affected, exhibit similar changes.

PARALYSIS.

When paralysis is due to a lesion of the spinal cord it is bilateral (*paraplegia*). Where there is a complete transverse lesion of the cord (transverse myelitis) there is motor and sensory paralysis of the parts below the site of the lesion, and in addition the *urethral* and anal sphincters are usually affected. If the cells of the anterior cornu are implicated, there is atrophy of the paralysed muscles, which show the reaction of degeneration. If these cells escape such atrophy as may exist is slight, and due merely to disuse of the affected limbs.

To understand the Diseases of the Spinal Cord, where many of the chronic degenerative lesions tend to involve one or more clearly defined nervous tracts, it is important to grasp what fibres are interrupted, or what cells destroyed, by lesions of these tracts. The student should therefore construct diagrams for himself, using those on p. 375 as a guide, and mark upon them the sites of various lesions in relation to the particular tracts involved. He will then be able to reason what must happen in any given instance. The lower diagram shows upon the left side the sites of typical lesions of one system only, upon the right the areas involved where more than one tract is implicated at the same time.

DISEASES OF THE SPINAL MEMBRANES.

Spinal meningitis or inflammation of the meninges may be either acute or chronic. When the dura mater is principally affected the disease is termed pachymeningitis; when the pia mater is most involved, leptomeningitis. This distinction is convenient when we have to deal with a *slow* process, as in *chronic meningitis*. It should, however, be remembered that in acute inflammation, though one or other membrane may be primarily affected, the disease quickly spreads, and involves the whole three membranes equally.

ACUTE SPINAL MENINGITIS.

The process may begin in the cellular tissue *outside* the dura mater—*i.e.*, external meningitis; or within the sheath, internal meningitis. The slight differences in the clinical symptoms will be easily understood if we consider the respective causes, and recall one or two physiological facts—

1. *Irritation* of nerve-roots leads to severe radiating pains and increased reflexes.

2. *Compression* of the cord substance causes paralysis, and loss of reflexes in the area of the compression; consequently, we are prepared for a greater degree of local excitability when the inflammation is external, and a more extensive area of the lesion in the internal meningitis, in which the cord *substance* will also be involved more quickly.

PACHYMENINGITIS
OR EXTERNAL MENINGITIS.

Causes.—

Extension of contiguous disease, such as—

1. Caries of spine.
2. Fracture of spine.
3. Tumours.

Pathology.—

1. The dura mater is red and injected, with loss of lustre.
2. Exudation of lymph.
3. Accumulation of pus between *spine* and *membrane*. The pus frequently dries and forms a caseous mass.

LEPTOMENINGITIS
OR INTERNAL MENINGITIS.

Causes.—

1. Extension of external meningitis on the one hand, or of myelitis on the other.
2. Extension of *cerebral* meningitis. Epidemic cerebro-spinal meningitis.
3. Septic state of blood, as in fevers, etc.
4. The inflammation may be tuberculous.

Pathology.—

The membranes are involved to a much wider extent. The internal membranes suffer early. The arachnoid is frequently wholly disorganised, the pia mater deeply injected, and the dura mater bulged *outwards* from accumulation of pus; in severe cases, the cord *substance* is *semi-fluid*.

PACHYMENINGITIS
OR EXTERNAL MENINGITIS.

Symptoms vary greatly.—

1. Severe cutaneous pains followed by anaesthesia.

2. Localised pain in the back, worse on movement or pressure.

3. Exaggerated reflexes till pus is formed, then paralysis sets in, first in the legs, then ascends, and reflexes become abolished. Anaesthesia replaces hyperaesthesia; sphincters are paralysed; septic fever and lividity of the skin supervene; large bed-sores form, and the patient often succumbs from exhaustion.

Treatment.—

1. Remove cause, if possible, by trephining, or aspiration of abscess cavity, etc.

2. Absolute rest.

3. Counter irritation, cupping, actual cautery.

4. Drugs—bromides, chloral, morphine. Ergot or atropine may be tried, to diminish the engorgement of the spinal circulation.

LEPTOMENINGITIS
OR INTERNAL MENINGITIS.

Symptoms.—

Are much the same, but of greater severity. Rigor and hyperpyrexia are frequent at the onset. There is more general excitement; opisthotonos, and retraction of the head are more marked; vasomotor centres are more deranged; and cerebral symptoms are much more common. Cheyne-Stokes breathing is often typically seen. There is marked cutaneous hyperaesthesia, and Kernig's sign may be present (see p. 43). Paralysis follows, with abolition of the reflexes. It may be impossible to distinguish this form from cerebro-spinal fever, or even acute myelitis, in which, however, pain is usually slight or absent.

Treatment.—

The same treatment, but Gowers advises in addition the free application of oleate of mercury, and internal administration of *calomel*. Both forms of meningitis are very little affected by drugs.

CHRONIC SPINAL MENINGITIS.

This is frequently the sequel to the acute affection, therefore dependent on the same causes; but it is sometimes chronic from the beginning. In these cases, syphilis, alcoholism, tubercle, or extension of any chronic disease of the cord are by far the most frequent causes. Severe chills, concussion, or traumatic lesions may act as exciting causes.

Pathology.—It varies much in different cases, from a mere thickening and cloudiness of the membrane with *increase* of cerebro-spinal fluid, to obliteration of the subarachnoidal space by complete organisation of the inflammatory lymph, which mats the pia mater and dura mater together.

Nerve-Roots.—At first are swollen and injected, but become fibrous and atrophied.

Spinal Cord suffers in proportion to—

1. The amount of compression from without.
2. The amount of thickening of the processes of the pia mater which *run into* the cord.

It should be noted that when there is *much fibrous* formation between the *dura mater* and the *cord substance* the condition is termed hypertrophic leptomeningitis.

Symptoms are those of local irritation of the nerve-roots, followed by paralysis; thus, from irritation of sensory fibres we get—

1. Pain in back with stiffness.
2. Sharp, darting, burning pains; occurring in various parts of the body, paroxysmal in character.
3. Spasm and rigidity are much less marked than in the acute form.
4. After the cord has become invaded the symptoms will be those of local myelitis.

Treatment.—Remove any cause if possible, then treat the myelitis.

DISEASES OF THE SPINAL CORD.

ACUTE MYELITIS.

A localised or extensive inflammation of the cord substance, affecting either the grey matter only, or grey and white together. The disease may be acute or subacute, the difference consisting largely in the rapidity of onset. The so-called

“chronic” forms are really more of the nature of degenerations than of inflammation.

Causes—

1. Extension of acute spinal or cerebral meningitis.
2. Exposure to cold, etc.
3. It may occur in the course of fevers, or other toxic states of the blood.
4. Concussion, especially when attended by fracture.
5. Disease of the vertebræ.

Morbid Anatomy.—The cord is swollen and softened at the affected part. It may be hæmorrhagic. The pia mater is injected. The meninges are occasionally involved.

Symptoms again are due (1) to irritation of nerve roots, and (2) to paralysis from implication of cord substance. The onset is rapid and usually accompanied by fever. The following is a good table of the results of a total transverse lesion *after paralysis has become established*:—

Above the lesion—

1. Reflexes, etc., normal.
2. Hyperæsthesia at upper margin of lesion, often accompanied by “girdle sensation.”

At the point of lesion—

1. Loss of reflexes.
2. Atrophy of muscles supplied by the implicated nerves.
3. Altered electrical conditions (*R. D.*).
4. Cutaneous insensibility.

Below the lesion—

1. Partial or total loss of sensation.
2. Complete paralysis (paraplegia).
3. Muscular rigidity or spasm, with exaggeration of tendon reflexes.
4. No alteration in trophic relations except from non-use. In the diffuse form where the grey matter is involved in a great part of its length there is rapid wasting of muscles.

5. *Vasomotor dilatation.*
6. *Rectal Centre.*—Unconsciousness of need, and inability to prevent evacuation; therefore, we get constipation; and, *incontinence of fæces after an aperient.*
7. *Vesical Centre.*—Again unconsciousness of need, and inability to prevent micturition; so we get reflex evacuation in gushes at intervals. Cystitis is frequent from decomposition of retained urine, retention being the rule at first, incontinence later.
8. *Sexual Centre.*—Absence of desire, but frequent reflex erections and emissions.
9. Ordinary reflexes are exaggerated; the slightest touch of the bedclothes, catheter, etc., causing severe spasm.

The course is variable. In cases that recover, sensation returns first, motor power much later, and usually imperfectly. The disease may extend upwards, causing respiratory paralysis and death, or death may follow bedsores or ascending nephritis.

Treatment.—Absolute rest on the side or in the prone position; leeches, dry cupping, or fomentations to the spine; ergot or belladonna to contract the spinal vessels; mercury. Prevent bedsores, relieve urinary retention. Later tonics, massage, and electricity to maintain the muscular nutrition.

INFANTILE PARALYSIS OR ANTERIOR POLIOMYELITIS ACUTA.

This, as the name implies, is an acute inflammation of the anterior cornua of grey matter.

“Infantile” is not a good term, for though infants and young children under eight are most often affected, adults sometimes develop the affection.

Causes.—Not known. “Chills” from exposure to wet and cold are usually blamed. The inflammation is limited to the

distribution of the median branch of the ventral spinal artery, and Marie therefore regarded it as due to thrombosis or embolism of the vessel. It is now generally considered to be of infective origin, though no special organism has been isolated. It begins like an infective disease, it is prevalent at certain seasons (summer and autumn), and it sometimes occurs epidemically.

Pathology.—*At first*—

1. Anterior grey horn is red and swollen.
2. Minute extravasations of blood occur.
3. Cloudy swelling and rapid destruction of anterior ganglion cells. Degeneration can be traced into the anterior roots.

Later—

4. The motor nerve trunks show marked changes, the fibres being smaller and fewer in number.

5. The “neuroglia” becomes increased, and the grey horn as a whole is *shrunk*. (See Sclerosis, page 384.)

Muscles are pale and flabby; atrophy begins early, and is well marked.

Microscopically, the changes already described (see p. 383) take place when muscles are cut off from trophic nerves.

1. Disappearance of striæ.
2. Atrophy of muscle cells, and
3. An increase of fibrous elements.

Symptoms.—First, reason what we might expect. We have an interference with the reflex loop, destruction of motor cells and motor trophic influence. The symptoms must be paralysis, absence of reflexes, and atrophy; the extent and severity of these symptoms will vary with the extent of the lesion. The onset is sudden, and after two or three days of pain in the limbs, feverishness, and sometimes vomiting or convulsions, the paralysis becomes established. It may be unilateral or bilateral, and is at first widespread, affecting usually the *lower* limbs, but all four limbs or only one, or only a *group of muscles* may be implicated. In a short time the paralysis passes away, except from a group or groups of muscles in which atrophy, loss

of reflexes, and reaction of degeneration, quickly supervene (see page 382). There is no rigidity or spasm. Note that muscle wasting may be concealed by fat.

The bladder and rectum usually escape. Sensation is but slightly affected, if at all.

Results.—

1. Nutrition of bones, etc., is seriously affected, and the child may recover motor power, with a wasted limb.

2. Permanent deformities produced by—

(1) Want of antagonism or unresisted contraction.

(2) Weight of foot, etc., causing extension, and the occurrence of ankylosis in that position.
(*Talipes equinus.*)

(3) Curvature of the spine may follow shortening of one leg.

3. Even in most favourable cases there is some slight permanent deviation from a perfectly normal muscular apparatus.

Treatment.—Rest, warmth to the affected part of the spine, fever diet, and laxatives.

Later.—Strychnine, cod-liver oil, lacto-phosphate of iron and lime, electricity, and massage.

Gowers emphasises that complete recovery is impossible after a year, though a gradual improvement may to a certain extent take place from increased *movements* on the part of the patient.

CHRONIC DISEASES OF SPINAL CORD.

Having described the more common acute affections, we will now take up the more common chronic affections of the cord. These are known as “system diseases,” by which is meant that in each particular disease one particular system of

neurons, or more than one, undergoes degenerative change. They may be classified as follows (Ormerod) :—

1. Degeneration of the Afferent Neurons: Posterior Sclerosis = *Locomotor Ataxy*.

2. Degeneration of the upper Efferent Neurons: Lateral Sclerosis = *Primary Spastic Paraplegia*.

3. Combined Degeneration of Afferent and Efferent Neurons: Postero-lateral Sclerosis = (1) *Ataxic Paraplegia*; (2) *Hereditary Ataxia*.

4. Degeneration of the lower Efferent Neurons = Progressive Muscular Atrophies of Spinal Origin. (1) *Chronic Anterior Poliomyelitis* or *Progressive Muscular Atrophy*, or (2) Combined with Degeneration of the upper Efferent Neuron = *Amyotrophic Lateral Sclerosis*.

LOCOMOTOR ATAXIA.

Definition.—A nervous disease characterised by an indefinite onset; chronic, but progressive course; and attended with marked symptoms of inco-ordination, trophic changes, and disturbances of special sense.

This disease is the best example of disease affecting the posterior columns—though it is, as will be seen, associated with sclerotic changes in the cerebrum, etc.

Ætiology.—Most common in middle-aged men. Syphilis, exposure to severe weather, etc., sexual excesses are the usual causes. The last two act as determining causes, but syphilis is most certainly a factor in producing this disease. It is, however, not *directly* a syphilitic lesion of the cord; in the great majority of instances it is uninfluenced by antisiphilitic treatment, and in rare cases it follows the action of other toxins. It is assumed to be a “parasyphilitic” disease, *i.e.* due to the circulation in the blood of a toxine produced by syphilis, and having a special action on nervous tissue. Gowers states that the influence of syphilis can be excluded with confidence in less than 10 per cent of all the cases.

Pathology.—Sclerotic changes in (see page 384) —

1. Posterior nerve roots, and posterior root ganglia.
2. Posterior columns—Burdach first, then by secondary degeneration into Goll's column.
3. Restiform body (medulla).
4. Optic thalamus.
5. Certain nuclei of cranial nerves.
6. The peripheral nerves.
7. When the disease has advanced, it may attack any area.

The disease begins as a rule in the lower levels of the cord, and extends upwards. Thus, below, the whole of the posterior columns are affected, while, above, the lesion may be limited to Goll's column.

Symptoms.—A careful digest of the foregoing pathological sites will at once show that the symptoms will vary much in each case, but we are at once prepared for sensory changes, inco-ordination, and changes in the sight apparatus.

The disease is usually divided into three stages—

1. Pre-ataxic.
2. Ataxic, or stage of inco-ordination.
3. Stage of Paralysis.

PRE-ATAXIC STAGE.

Symptoms are most insidious. Taking a typical case we shall get—

1. *Changes due to irritation of sensory roots*—

- (1) Lightning pains over the body : hot, burning, and tingling in character ; lasting a few seconds. They usually begin in the lower limbs.
- (2) Girdle pains (from the upper margin of sclerosis), the patient complaining of constriction, as if an iron band was around him.

2. Early loss of knee-jerk.

3. Symptoms in connection with the optical apparatus—

(1) Argyll-Robertson pupil (reflex iridoplegia).

The pupil fails to react to light, but accommodation remains unaffected.

(2) Diplopia or double vision.

(3) Primary optic atrophy.

(4) Paralysis of ocular muscles.

(5) Extreme contraction of the pupils (miosis).

} May occur early
or late.

4. It is alleged that sexual capacity is increased in the early stages; *later, sexual desire is abolished.*

5. Not infrequently imperfect control of micturition, or retention.

ATAXIC STAGE.

Sometimes the first sign of inco-ordination is tumbling forward into the basin on closing the eyes during the morning wash, but once begun the inco-ordination often rapidly develops into the characteristic "Ataxic gait." The patient feels and is unsteady on turning round or standing with his eyes shut, and fails to walk on a straight line, etc. (See Gaits.)

Romberg's sign is the usual test for ataxia. If the patient is made to stand with his feet together (heels and toes), and to close his eyes, he sways from side to side, and may fall if not supported.

Anæsthesia of the soles of the feet soon comes on. There is complaint of numbness, or of a sensation of walking on wool or indiarubber. The anæsthesia may be tactile, thermal, muscular, or painful. Thus the patient may fail to distinguish with his feet the difference between a hot and cold body, or between heavy and light weights, provided they are similar in appearance.

Ataxia may affect the hands as well as the feet, making it difficult to unfasten the clothing, etc.

It should be noted particularly that the muscular *power* is not diminished, for the sufferer can resist movement or push away a heavy weight.

Often at this period, occur peculiar visceral disturbances, termed crises. The best-known are—

1. Gastric crisis—intense epigastric pain, hyperacidity, and vomiting.

2. Laryngeal crisis—noisy stridulous breathing, with great dyspnoea. This is, however, rare.

3. Vesical crisis—paroxysmal retention of urine.

4. Rectal crisis—tenesmus, etc.

The other most marked changes are the so-called “trophic” alterations :—

1. Skin becomes dry, or shiny and glossy with absence of hair ; nails crack, etc.

2. *Joints*.—Charcot’s disease (tabetic arthropathy). The cartilages are eroded, the ends of the bones wasted, and the ligaments ossified. There may or may not be effusion into the joint.

3. Perforating ulcer of foot.

4. Brittleness of bones, ulceration of cartilage, etc.

PARALYTIC STAGE.

The patient becomes bed-ridden, and liable to grave inter-current diseases ; and hemiplegia, pneumonia, or gangrene, etc., ushers in a fatal ending. Vesical troubles are aggravated ; cystitis may set in, and end in an ascending pyelo-nephritis.

It must be particularly noted that some cases run an extremely lengthened, others a very rapid course, and though it is convenient to divide the disease into three stages, there is no hard-and-fast line or distinct margin between them.

Treatment will be considered under Treatment of Chronic Diseases of the Spinal Cord (see p. 405).

PRIMARY SPASTIC PARAPLEGIA.

(PRIMARY LATERAL SCLEROSIS.)

Primary spastic paraplegia is a paralysis attended with spasm and rigidity, resulting from sclerosis of the anterior pyramidal and crossed pyramidal tracts.

Let us consider what we must expect from interruption of these two tracts.

1. *The brain being cut off from the motor nerves two conditions are brought about :—*

- (1) *Voluntary* motion must be imperfect, according to the extent of lesion.
- (2) Reflexes must also be exaggerated, as they are cut off from the inhibitive influence of the brain, but the reflex loops are not interfered with. (See Diagram, page 375.)

2. *The motor tracts, being cut off from the first trophic realm in the cortex*, will undergo secondary degeneration, which causes at first *increased irritability*, which will excite *motor roots* and cause spasm and rigidity.

3. The anterior horn not being affected, there will be no interference with the second trophic realm, and consequently no atrophy of muscles.

Ætiology.—The disease is comparatively rare, but is most common in males between the ages of twenty and forty. It is sometimes congenital, owing to bilateral meningeal hæmorrhage over the central convolutions following injury during birth.

Symptoms.—

1. Weakness and stiffness of the lower limbs.
2. Exaggerated knee-jerk, and presence of ankle clonus.

Babinski's sign is generally present, *i.e.* when the sole of the foot is stroked by a pointed instrument, the toes, especially the great toe, are *over-extended* towards the dorsum of the foot, and abducted one from the other.

3. Spasm and rigidity causing forcible *adduction* and extension of the limbs, rendering the gait characteristic. (See Gaits.)

The rigidity is nearly continuous, and when relaxations take place the slightest stimulus causes spasm.

Though the legs are the limbs usually attacked, still the

muscles of the trunk, and the arms occasionally, may be first involved.

The disease runs an extremely chronic course, as, until it becomes widely spread, the muscles remain plump, sensation unaffected, and sphincters unimpaired. Finally, there may be complete paralysis of the affected parts, or the lesion may extend to other tracts of the cord.

Differential Diagnosis.—(See Table, p. 404).

Treatment is considered under Treatment of Chronic Diseases of the Spinal Cord.

PROGRESSIVE MUSCULAR ATROPHY.

Progressive muscular atrophy, or chronic anterior poliomyelitis, is *the* example of a *chronic* affection of the anterior grey horn and motor roots. In this instance, as in the acute affection, the site of the lesion leads us to expect disturbances of reflexes, atrophy, and deficient muscular power; but the sclerotic changes being more localised and of slow growth, the clinical symptoms will vary.

Ætiology.—Males are more often affected than females. The disease is one of adult life. It is often associated with exposure to cold and wet, but is not limited to the labouring classes. Often no cause can be found.

Pathology.—Sclerosis of anterior horn, anterior roots and nerve trunks, with changes in groups of muscles.

1. Anterior grey horns are pale, but not altered much in shape.
2. Great increase of neuroglia.
3. Obliteration of nerve cells.
4. In the white substance, sclerosis of the antero-lateral tract.

Anterior nerve roots are markedly atrophied.

Nerve Trunks.—Changes are not so marked as in the roots, for the sensory fibres in the mixed nerve trunk are unaffected, and frequently *some* of the motor fibres escape.

Muscles. —Pale, flabby, etc., as in the acute affection; but fatty, vitreous, or waxy changes are also present. They exhibit the reaction of degeneration in proportion to the amount of wasting. It may be absent in many instances owing to the escape of some of the fibres of the motor nerve, so that degenerated and healthy muscle fibres are intermixed. There is then diminished irritability, both to faradism and galvanism.

Symptoms.—The disease usually begins in one or both of the upper extremities with atrophy of—

1. Thenar and hypothenar eminences.
2. Interossei.
3. Forearm muscles, and those of the shoulder.

Paralysis of the interossei, with unopposed action of their opponents, produces the claw-like hand (*main en griffe*)—i.e., the first phalanges are *hyper-extended*, the middle and distal phalanges are flexed on the first phalanges. The muscles of the back are early affected, but the upper third of the trapezius escapes until quite late. After this, the extension is extremely gradual, and years may pass before both arms, both legs, intercostals, or diaphragm are attacked. The legs are usually affected late, but the disease may commence in them. The affected muscles generally exhibit the peculiar fibrillary twitchings of dying or exhausted muscle. These twitches can be readily produced by a gentle tapping; they are often visible to the patient, lasting two or three minutes, and starting without any apparent stimulus. The disease may cease spontaneously, usually at a late stage, or may end fatally from complication with bulbar paralysis or intercurrent pneumonia, etc.

Treatment will be summed up under Treatment of Chronic Diseases of the Spinal Cord.

Differential Diagnosis. (See Table.)

CHRONIC SPINAL DISEASES,

With mixed Lesions or "Overflows."

We have, in the foregoing, studied total and partial lesions ; and found that in the

Posterior affection—inco-ordination and anæsthesia were the marked features.

Lateral affection—spasm and rigidity with increased reflexes.

Anterior affection—atrophy and loss of reflexes were the prominent symptoms.

It is easy to see that we might get compounds of these ; for instance, we might have an—

Antero-lateral lesion = incomplete atrophy with spasm ; or

Postero-lateral = inco-ordination and spasm.

Indeed, it would seem more probable at first sight to get these "mixed" conditions than isolated or limited lesions.

We shall next describe the three best-known mixed lesions, viz. :—

1. Postero-lateral sclerosis or Ataxic paraplegia.
2. Hereditary Ataxia.
3. Amyotrophic lateral sclerosis, in which both the upper and lower efferent neurons are involved.

ATAXIC PARAPLEGIA.

Ætiology.—Occurs most frequently in males, beginning in early middle age. There is rarely any connection with syphilis, and the ætiology is largely conjectural. Exposure to cold, sexual excess, etc., are blamed, as in all chronic spinal affections.

Pathology.—Sclerosis of—

1. Lateral columns (direct and crossed pyramidal tracts, and direct cerebellar tracts).
2. Posterior columns (but not the roots). The thoracic part of the cord is more affected than the lumbar.

Symptoms.—Obscure in the early stages.

1. Patient complains of tiredness and weakness of the limbs (*no lightning pains*).

2. Inco-ordination, patient reels or sways if the eyes be closed, and he fails to walk on a straight chalked line.

3. *Increase* of knee-jerk (ankle clonus is often present). Babinski's sign may be present.

4. As the disease progresses the tendency is to become *more spastic*, and less ataxic in character—*i.e.*, inco-ordination does not increase, but *spasm and rigidity* become markedly increased. Both ataxy and spastic weakness may extend to the upper limbs.

It should be noted that there is no anæsthesia, lightning pain, or alteration of the optical apparatus; points which distinguish it from locomotor ataxia; and this is what we should expect, seeing there is no involvement of the posterior *nerve roots, or cranial nerves*.

FRIEDREICH'S DISEASE (Hereditary Ataxia).

Ætiology.—The disease may or may not be hereditary. It is strictly rather a family disease, tending to affect several brothers or sisters. It begins as a rule in childhood or about the age of puberty.

Pathology.—Sclerosis involving principally the “neuroglia” element in—

1. Lateral columns—The direct cerebellar tract, the comma tract of Gowers, and part of the crossed pyramidal tract.
2. Posterior columns.
3. Clarke's columns in grey matter (to a slight degree).
4. Certain cranial nuclei.

Symptoms.—

1. Inco-ordination of a jerky kind of the lower extremities.
2. Inco-ordination of the *arms* (somewhat later).
3. Hesitation in speaking, or a jerky manner in delivery of speech.

4. Unsteadiness of the *head, and oscillatory movements of the eyeballs* (nystagmus).

5. Impairment of sensation—the anæsthesia is not marked in most cases.

6. Absence of knee-jerk.

7. *Talipes equinus and other deformities*, especially curvatures of the spine.

Note *absence* of lightning pains, trophic changes, and the Argyll-Robertson pupil to distinguish this disease from locomotor ataxia. The marked inco-ordination and nystagmus render the diagnosis from spastic paraplegia easy.

AMYOTROPHIC LATERAL SCLEROSIS.

Pathology.—Sclerotic degeneration of—

1. Crossed pyramidal tracts, extending upwards sometimes to the pons and medulla, sometimes even to the cortex.
2. Cells of the anterior cornua, and, consecutively, the fibres passing from these to the muscles.

Ætiology, beyond that common to all chronic diseases of the cord, is practically unknown. Syphilis is not a factor. The age of onset is from twenty-five to fifty. Both sexes are liable.

Symptoms.—

1. Weakness and wasting of the *upper* extremities coming on very slowly. Pain and disordered sensation may be present, but not anæsthesia. Electrical excitability of the muscles is lessened for both galvanic and faradic stimuli.

2. *Increase of reflexes with spasm* (until the wasting is profound; then, the anterior multipolar cells being extensively diseased, the reflexes *may be actually abolished*). The spasm and rigidity cause a peculiar deformity which is characteristic, viz. :—

Arm extended close to the body.

Forearm semi-flexed and pronated.

Wrist strongly flexed.

Fingers bent into the palms. (CHARCOT.)

3. Later, in the second stage, spastic paralysis commences in the *lower extremities*. It is accompanied by exaggeration of the reflexes, and followed by atrophy. The sphincters are not affected.

4. Finally, in the third stage, there occur symptoms dependent on *extension of the disease to the motor nuclei of the medulla*. The tongue or lips may be affected, the palate paralysed, the speech nasal. Dysphagia may follow. Septic pneumonia, or cardiac or respiratory paralysis, may end the scene.

The disease runs a quicker course than the chronic lesions of the cord already described, and death usually occurs in from one to three years.

The beginner is frequently puzzled to understand why, in this disease where *the anterior horn* is involved, reflexes are increased instead of lost. The explanation is, that this affection *begins*, in many cases, in the "*lateral*" region (upper efferent neuron), but *extends* into the anterior horn (lower neuron), paralysing only a certain number of motor cells, and *irritating* others.

Although this disease was described by Charcot as distinct from progressive muscular atrophy, it is evident that the lesions in the cord are practically identical, the pyramidal fibres being affected in both cases. "The difference between them is that in the one case there is degeneration only of the lower spinal motor neuron, in the other there is also decay of the cerebral neuron, which comprises the cortical cell and pyramidal fibre" (Gowers). There are many cases which present intermediate stages between typical progressive muscular atrophy and typical amyotrophic lateral sclerosis, these two forms representing "extreme examples at the opposite ends of a series."

Differential Diagnosis of Chronic Diseases of the Spinal Cord.

TABLE I.

	LOCOMOTOR ATAXIA.	ATAXIC PARAPLEGIA.	FRIEDREICH'S ATAXIA.
<i>Age</i>	Middle-aged men.	Early middle life; males.	Childhood or early youth.
<i>Causes</i>	The toxic effects of syphilis, rarely other toxins.	Exposure to cold, traumatisms, etc.	Occurs in many of the same generation. Neurotic predisposition.
<i>Ocular symptoms</i>	Various muscular paralyses or palsies.		
{ <i>Argyll-Robertson pupil</i>	Present.	Absent.	Absent.
{ <i>Nystagmus</i>	Absent.	Absent.	Present.
<i>Tendon reflexes (knee-jerk)</i>	Lost	Increased.	Lost.
<i>Disorders of Sensation</i>	Lightning pains prominent; girdle sensation; numbness of feet.	Absent.	Absent usually; occasional paræsthesiæ.
<i>Inco-ordination</i>	Characteristic gait; lower limbs chiefly affected, upper limbs later.	Ataxia marked; spasm and rigidity also present, and tend to increase.	Marked, but irregular and jerky; may affect upper limbs.
<i>Speech</i>	Unaffected.	Seldom affected.	Often affected.

TABLE II.

	PROGRESSIVE MUSCULAR ATROPHY.	AMYOTROPHIC PARALYSIS.	PRIMARY SPASTIC PARAPLEGIA (LATERAL SCLEROSIS).
<i>Limbs most affected</i>	Upper — atrophy begins in thenar and hypothenar eminences. Unilateral at first.	Upper — atrophy may begin in muscles of forearm or deltoid. Unilateral.	Lower— <i>no atrophy</i> ; but rigidity and spasm are present. Bilateral.
<i>Deformity</i>	The "claw-like" hand.	Flexion of elbow, pronation of hand, flexion of wrists, and fingers into palms.	Adduction of legs. They may cross each other.
<i>Tendon reflexes (knee)</i>	Unaffected.	Unaffected.	Exaggerated on both sides.
<i>Electrical changes</i>	Reaction of degeneration sometimes present.	Partial R. D. or diminished excitability.	Normal as a rule.

It must be remembered that progressive muscular atrophy and amyotrophic paralysis may merge the one into the other.

GAITS.

In the various forms of chronic disease of the spinal cord, whether the lesion be confined to the tracts in the cord itself or more extensive, there are certain peculiarities of gait which help to create a typical picture of the disease. These may, therefore, be briefly characterised in this place.—

1. *The Ataxic Gait.*—In locomotor ataxia, as the patient walks, his feet are separated from each other, his heels are brought down first in a stamping fashion, he watches his feet to correct by sight the imperfect muscular sense, and more power is used than is necessary for progression, so that the feet are, as it were, flung out beyond the line of progress. If he stands with the feet together he tends to sway; if he tries to turn sharply he may reel or fall.

2. *The Spastic Gait.*—In lesions such as spastic paraplegia, while the patient can still walk, he drags the rearward leg slowly forward, the toes scraping the ground. The knee and ankle are very slightly flexed, and hence there may be “circumduction.” There is often strong adductor spasm, so that the feet may cross each other. When the spastic element is prominent, the gait may assume a “hopping” character.

In *disseminated sclerosis* (*q.v.*), besides the spastic gait, there may be tremor on exertion (“intention tremor”).

TREATMENT OF CHRONIC DISEASES OF THE SPINAL CORD.

The same general principles apply in the treatment of all forms of chronic nervous disease. The general strength must be supported, and the mode of life carefully regulated. Sexual or alcoholic excess is to be strictly avoided. Every effort is to be made to ensure sound sleep. Electrical treatment, while

sometimes of temporary benefit, has no real influence on the course of the disease. It must be remembered that apparent improvements are often due, not to treatment, but to temporary remissions or intermissions. Electricity is, however, useful in maintaining or restoring the nutrition of partially atrophied muscles, as in infantile paralysis.

Few drugs have much effect upon the process of sclerosis. Most reliance is to be placed on strychnine, which may be given either by the mouth or hypodermically, and by preference in the latter method. It should, however, be used with caution in spastic conditions, from its tendency to increase the spasm. In these circumstances arsenic is often of more service. The close connection of locomotor ataxia and syphilis would suggest specific treatment, but it is generally useless. The only cases which may benefit from it are those where the ataxia has rapidly followed the initial lesion (two years or so), and where the case is seen early. The lightning pains and visceral crises of this disease may require the use of morphia, but they are better treated as a rule by anti-neuralgic remedies such as phenacetin, antipyrin or aspirin. Chloride of aluminium (four grains thrice daily) is also said to be useful in this regard. The ataxy may be treated by Fraenkel's system of re-educating the muscles, beginning with very simple exercises, and proceeding to more complicated manœuvres as control is regained. Massage is also of considerable use.

MYOPATHIES.

PROGRESSIVE MUSCULAR DYSTROPHY.

PSEUDO-HYPERTROPHIC PARALYSIS.

Progressive wasting of various groups of muscles, with or without initial hypertrophy. The primary change is in the muscles themselves.

Ætiology.—Occurs principally amongst boys; and there is a tendency to affect more than one male member in the same family. It is hereditary in a peculiar manner, as whilst it

affects males, it is transmitted through the females—*i.e.*, in a certain family a son was affected ; his sister married, and had two daughters and one son ; the daughters escaped, but the *boy* was affected. This hereditary tendency is the only known etiological factor, but in 44 per cent of Erb's cases it was absent. The disease commonly begins in childhood.

Pathology.—The essential changes seem to be in the muscular tissues alone. Changes in the cord are rare, and probably are only accidental complications. Microscopically there are seen in the affected muscles—

1. Increase of fibrous tissue.
2. Atrophy of muscle fibres, possibly preceded by hypertrophy.
3. Large deposit of fat.

The enlarged appearance of the muscles is due more to fat than the increase of fibrous tissue. The muscles mostly involved are as follows—

ENLARGED.	WASTED.
1. Calves.	1. Latissimus dorsi.
2. Glutei.	2. Teres major and biceps.
3. Infra-spinatus and deltoid.	3. Lower part of pectoralis major.

The above table of course is only approximate ; there seems to be no hard-and-fast line as to what muscles will show most changes.

There is no reaction of degeneration. The response to both galvanism and faradism becomes progressively less, but as long as any true muscular tissue is left there will be response to stimulation. The knee-jerk is lost when the tonicity of the extensors fails.

It is only one form of progressive muscular dystrophy that is accompanied by hypertrophy, or pseudo-hypertrophy. Others may from the first be attended by atrophy. The disease, too, does not always begin in childhood, but sometimes

in adolescence, or in early adult life. Erb distinguishes the following forms :—

I. In children.

1. Hypertrophic form.

(a) With pseudo-hypertrophy.

(b) With real hypertrophy.

2. Atrophic form.

(a) With primary involvement of the face.

(b) Without such involvement.

II. Progressive muscular dystrophy of youths or adults.

Symptoms are very characteristic when the disease is fully developed, but in the earlier periods they are somewhat obscure.

1. Impaired locomotion from muscular weakness ; the little fellow lags behind his playfellows.

2. Hypertrophy of muscles with atrophy of others.

3. Certain deformities, through contraction of muscles un-antagonised by the paralysed ones.

4. The characteristic gait and movements.

During walking the abdomen is thrown out, with a corresponding *hollowing of the back* ; the legs are widely spread out, the whole effect being a *waddling gait*.

The getting up from a recumbent position is most characteristic, especially if there be no objects near, by which the child can aid himself. He first gets on his hands and knees ; then extends his knees to the utmost ; and lastly, by a climbing movement—*i.e.*, grasping the knees with his hands alternately, moves higher and higher up the thigh till the trunk is raised.

It is remarkable how easily some patients can manage these movements ; later, the muscular weakness is such that they require the aid of near objects to grasp at, but finally failure is complete, the patient being helpless.

Prognosis in progressive muscular dystrophy is very unfavourable. Few patients survive to the age of 20, and the majority die of intercurrent pulmonary diseases, due to weakness of the respiratory muscles.

Treatment.—Expect most benefit from a thorough system of continued massage, well-planned gymnastic exercises, careful hygiene, and galvanism. The diet should be highly nourishing. Cod-liver oil or malt may be given.

Tonics such as strychnine and phosphorus may be given. Strychnine is the drug most frequently used, and it is best given hypodermically. Arsenic also is often used, and may be given hypodermically or by the mouth. The thinness of the chest walls renders the patient very prone to chest affections, which, should they occur, often carry him off, and are to be particularly guarded against.

THOMSEN'S DISEASE.

(MYOTONIA CONGENITA.)

This is a rare condition characterised by a tendency of the muscles to tonic spasm during attempts at voluntary movements.

The disease is probably always associated with some congenital defect. Heredity is a strong and important factor, and more than one member of a family may be affected.

Its pathology is unknown. The muscles are usually well developed as far as appearance goes; indeed, their fibres are often hypertrophied.

The Electrical Reactions are markedly disturbed. There is increased irritability, and the contraction following faradic, and especially galvanic, stimuli is unduly prolonged. A. C. C. is as easily elicited as K. C. C. A continuous galvanic current sets up a series of wave-like contractions passing from cathode to anode.

Symptoms.—

1. After *rest* the patient experiences tension and stiffness of the muscles on attempting to rise, etc.
2. Difficulty in *relaxing* the muscles.
3. In severe cases the spasm may be at first so great as to cause the patient to remain locked in the position in which he is; but it yields gradually to repeated attempts at movement.

4. The muscles of the tongue, face, and eyes may also be similarly affected. The acts of micturition, defæcation, and respiration are not affected.

Note that continued movements and warmth decrease the spasm; mental worry and cold usually aggravate it.

Treatment.—Massage, gymnastic exercises, electricity, and warm baths. No treatment has any real effect.

DISEASES OF THE MEDULLA.

Much that has been said about the spinal cord may be also said about the medulla, or bulb. It is a conductor of impulses to and from the brain, and it also contains the principal reflex centres. The more important points may be summed up as follows:—

1. It contains the deep origin of all the cranial nerves after the fourth. The nuclei of motor cranial nerves are homologous with the spinal motor nuclei in the anterior cornua, and may be affected simultaneously.

2. The *motor fibres* decussate to form the crossed and direct pyramidal tracts in the cord.

3. The *sensory* tracts become re-arranged as sensory fibres, prior to their course to the cortex.

4. It contains the following centres:—

(a) *Centres essential to life.*

1. Respiratory centres.
2. Cardiac centres—motor and inhibitory.
3. Vasomotor centres.

(b) *Centres connected with the alimentary canal.*

1. Centre for sucking.
2. Centre for mastication.
3. Centre for vomiting.
4. Centre for deglutition.

(c) *Centres connected with the eye.*

1. Centre for winking.
2. Centre for dilator pupillæ.

(d) Centres for secretion.

1. Salivary centre.
2. Lachrymal centre.
3. Sweat centre.

It will be seen from the above table that even a small lesion may be attended by grave and diverse symptoms. An extensive lesion would be incompatible with life. Some authorities doubt if the medulla is ever diseased primarily without a speedily fatal issue; it is often, however, affected in the later stages of spinal or basal disease, disseminated sclerosis, etc. Probably the best example of disease of the medulla is the so-called chronic progressive bulbar paralysis, often termed glosso-labio-laryngeal paralysis.

GLOSSO-LABIO-LARYNGEAL PARALYSIS.

An affection characterised by progressive paralysis and atrophy of the tongue, lips, etc., accompanied by difficult articulation and deglutition, ending in suffocation or inanition.

Ætiology.—It occurs most frequently in old people, and is rarely seen in persons under forty years of age. Men are more frequently attacked than women. Exposure to cold, blows and injuries to the neck, are usually put down as the principal causes. Often this disease is associated with chronic diseases of the spinal cord (progressive muscular atrophy or amyotrophic lateral sclerosis).

Pathology.—

1. *Sclerotic* changes are found in the—
 - (1) Motor nerve *nuclei* of the medulla.
 - (2) Motor nerve *roots* and fibres directly connected with the bulb.
2. Degenerative changes are found in the trunks and motor endings of the glosso-pharyngeal, spinal accessory, and hypoglossal nerves. There is atrophy of the tongue, lips, and muscles supplied by the above nerves. The muscular fibres are in a state of fatty degeneration, or may be replaced by fat.

The lesions originate in the motor nerve nuclei, the cells of which show shrivelled processes, shrunken cell bodies, absence of Nissl's bodies, and shrinkage or absence of the nuclei. The total number of cells is lessened. The changes extend throughout the neuron to the nerve-endings. The white substance of the medulla is often unaffected, but it may also undergo change.

Symptoms.—Obviously, with lesions so widespread the symptoms must vary with the nuclei involved ; and if spinal or basal disease be present, the symptoms of these will be super-added. Yet, in spite of these facts, a fairly typical picture of glosso-labio-laryngeal paralysis may be presented. Tabulated, the principal symptoms are—

1. *Impairment of articulation (dysarthria)*, due at first to defective movements of the tongue, but later, to the atrophy of the lips ; consequently, defective pronunciation of the letters involving the tip of the tongue is first most marked—*i.e.*, the letters T, K, D, or the exclamation SH ! ; later there is difficulty in pronouncing the letters U, O, OO, W, P, B, M, owing to implication of the lips. Finally, *speech may become unintelligible*, but phonation is rarely entirely lost.

2. *Difficulty in swallowing*. The food may accumulate between the lips and gums.

3. *Symptoms indicative of paralysis of the palate—i.e.*, nasal twang of voice, regurgitation of fluids through the nose.

4. *Dribbling of saliva from the mouth*. This is a most marked symptom, and the patient is continually wiping the secretion away. Possibly more saliva is secreted than normally, but this symptom may be due to deficient deglutition, so that saliva is not *swallowed*.

5. *Symptoms indicative of paralysis of the larynx* are lowering of the pitch of voice, aphonia, imperfect cough, etc. When the superior laryngeal nerve is paralysed, particles of food get into the lung and excite a fatal pneumonia.

6. *Symptoms indicative of paralysis of the cardiac centre* are paroxysmal attacks of dyspnœa, a sensation of tightness across the chest, tachycardia and irregular action of heart, etc.

The patient has usually a most sad expression, which is a contrast to his often buoyant spirits. There is impairment of emotional control. Tears or laughter are readily excited, though the intellect is unaffected. The condition of the tongue is very characteristic when the disease is well marked. Its muscular tissue is much atrophied, and the mucous membrane hangs in sack-like folds, wrinkled and covered with a dirty yellowish fur. It is tremulous, lies helpless in the mouth, and is only moved with great difficulty, hence the collecting of food between teeth and cheeks. Sometimes, however, the tongue may be broad and flabby, from the accumulation of interstitial fat. The muscles of mastication may be also involved (showing implication of the fifth nerve).

The electrical reactions are usually little altered, but R. D. is sometimes present in the affected muscles.

Prognosis.—Invariably fatal. Death may occur from emaciation, but is more often due to the various complications that arise, especially pneumonia.

Treatment.—Careful attention to all the details of health, with the administration of tonics such as arsenic or strychnine, are the usual measures. Electricity has been tried, without much benefit. The greatest care is essential in feeding, to avoid passage of food into the larynx. Feeding by the œsophageal tube may be required.

Acute Bulbar Paralysis of the apoplectic type is of vascular origin, and most frequently follows thrombosis. It occurs in old people with marked atheroma, but may follow syphilitic endarteritis in the young. Its onset is sudden, being accompanied by vomiting, vertigo, and sometimes loss of consciousness. There follow partial or complete loss of articulation (anarthria, *not* aphasia); dysphagia, with regurgitation of fluids through the nose; paralysis of the lower half of the face; and paralysis of the tongue, larynx, and pharynx. The paralysis may extend to the limbs, when the tendon reflexes are exaggerated, and Babinski's sign is found. Dyspnœa or Cheyne-

Stokes breathing may occur ; the pulse may be rapid, and the temperature raised. In fatal cases death may ensue in a few days, weeks, or months.

Treatment is mainly palliative,—attention to feeding, etc. In cases where syphilis is even suspected, energetic specific treatment should be undertaken, and may bring about partial recovery.

DISEASES OF THE BRAIN.

BEFORE beginning the study of cerebral disease we shall first consider a few of the elementary anatomical and physiological facts ; without a knowledge of such facts brain diseases are quite incomprehensible.

The brain proper consists of two large hemispheres, partly separated from each other by the great longitudinal fissure ; but bound together below by various commissures.

Externally, each hemisphere is covered with grey matter, named the cortex, which is mapped out by furrows into a series of folds, termed convolutions. These convolutions are of the highest importance ; absent in the lower type of animals, they become gradually more marked in the higher animal, well marked in monkeys, but only seen in perfection in the *highly educated adult human brain*.

Of the *Fissures* of the hemispheres the largest and most evident subdivide the surface of the cerebrum into lobes, and may be called *interlobar* ; the smallest fissures—*intralobar*—divide the lobes into convolutions, which have received definite designations.

The *Interlobar Fissures* are—

1. The *fissure of Sylvius*.
2. The *fissure of Rolando*.
3. The *parieto-occipital fissure*.

The *Lobes* of the cerebrum are five in number ; four are bounded by the interlobar fissures, and take their names from the bones of the skull in relation to which they lie.

They are—

1. The *frontal*.
2. The *parietal*.
3. The *occipital*.
4. The *temporo-sphenoidal*.

The fifth lobe—the *central lobe*—(*insula or island of Reil*) is not in contact with the bones of the skull, *but is hidden within the fissure of Sylvius*, the margins of which must be separated in order to see it.

The *external* convolutions of these lobes are—

1. Of the Frontal lobe—

Ascending	} Convolutions.
Superior	
Middle	
Inferior (or Broca's)	

2. Of the Parietal lobe—

Ascending parietal	} Convolutions.
Superior parietal	

3. Of the Occipital lobe—

Superior	} Convolutions.
Middle	
Inferior	

4. Of the Temporo-sphenoidal—

Superior	} Convolutions.
Middle	
Inferior	

The convolutions seen on the *mesial* surface of the brain are—

Gyrus fornicatus.
 Marginal.
 Hippocampal (uncinate).
 Dentate.
 Quadrangle (Præcuneus).
 Cuneus.
 Paracentral lobule.

INTERIOR OF CEREBRUM.

Internally the cerebrum consists of white matter, and ganglionic masses of grey matter. The solid mass of white matter *above* the corpus callosum is termed the *centrum ovale*.

Below the corpus callosum is an irregular and somewhat T-shaped cavity, divided by septa into smaller spaces, termed ventricles. The ventricles communicate with each other by variously named canals—thus, the lateral ventricles communicate with each other and the third ventricle, by the foramen of Monro; the third and the fourth ventricles by the “iter” or aqueduct of Sylvius; the fourth ventricle and the sub-arachnoidal space by the foramen of Majendie.

The *Basal Ganglia* are masses of grey matter situated at the base of the brain, viz.—

1. The *Corpora Striata*, consisting of two portions—

(1) Caudate nucleus = intra-ventricular portion.

(2) Lenticular nucleus = extra-ventricular portion.

2. *Optic Thalami*, containing sensory and optic fibres. The *upper* part of each thalamus appears in the lateral ventricles. The *under* surface rests upon the crura cerebri. (Between the lenticular nucleus externally, and the caudate nucleus and optic thalamus internally, lies the mass of white matter termed the internal capsule. See page 379.)

3. *Clastrum* is a narrow band of grey matter outside the lenticular nucleus. Function unknown. (See page 379.)

4. *Corpora Geniculata* are masses of grey matter forming rounded swellings on the lateral and median portions of the optic tracts.

5. *Corpora Quadrigemina* contain sensory fibres, and are also implicated in the movements of the eye.

6. *Pineal Gland*. Function unknown.

BLOOD SUPPLY OF THE BRAIN.

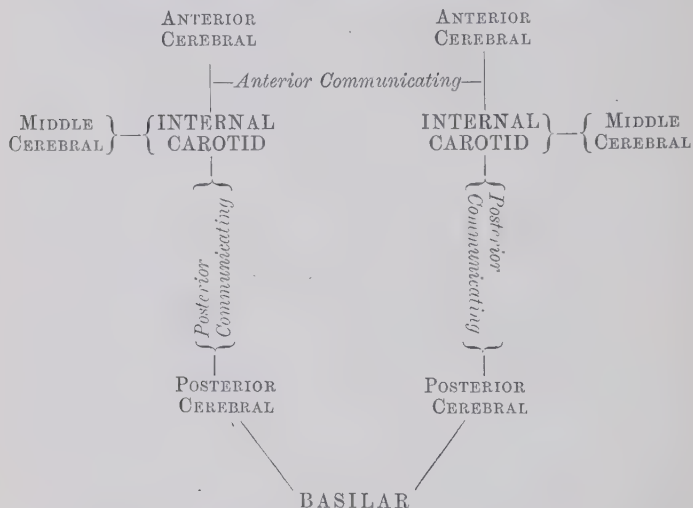
The arteries of the brain are derived from the two internal carotids, and the two vertebral arteries, which unite to form the basilar artery.

The branches are practically arranged in two sets, viz. :—

The Cortical group—(anterior, middle, and posterior cerebral arteries).

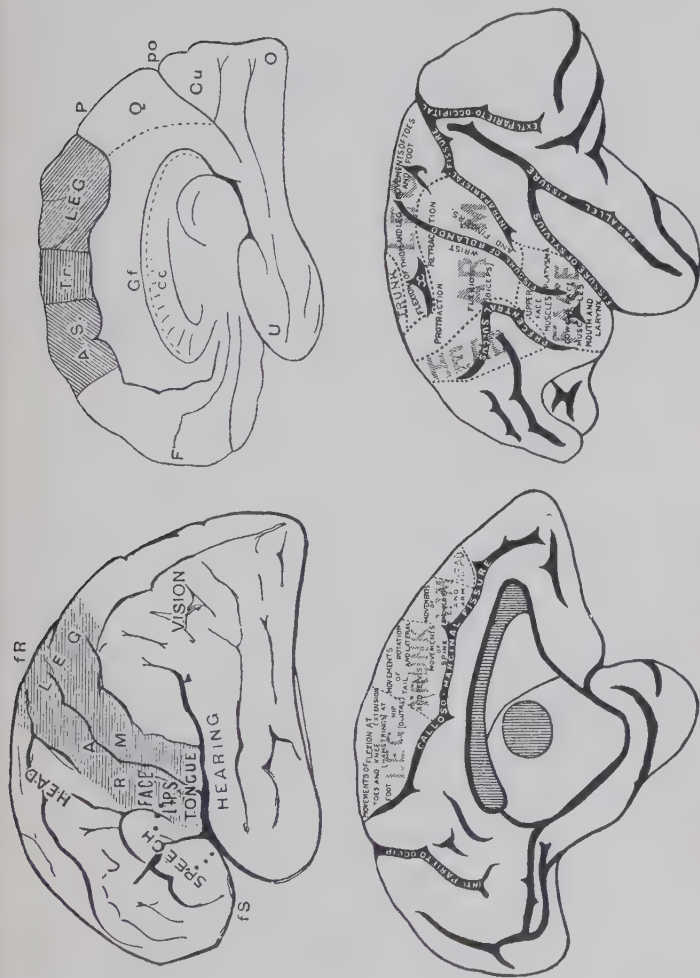
The Basal group—(comprising the circle of Willis, and the central arteries passing from it).

The circle of Willis may be represented by a diagram :—



So many pathological conditions result from cerebral hæmorrhage, that special attention should be directed to the course of the middle cerebral artery, branches of which so often rupture, that it is really *the* artery of hæmorrhage.

Middle Cerebral or Sylvian Arteries are the largest branches of the internal carotid, and pass upwards and outwards in the fissure of Sylvius till they reach the surface of the island of Reil, where they ramify in the pia mater, forming part of the cortical system of arteries. These branches supply the under and outer surface of the frontal lobe (in part), and the ascending frontal convolution, the outer surface of the parietal lobe, and most of the outer surface of the temporal lobe. They



DIAGRAMS showing the Principal Centres in the Brain (after Horsley and Beevor).

anastomose freely with the anterior and posterior cerebral arteries. Other branches, furnished through the anterior perforated spot to the corpus striatum, are all terminal arteries and belong to the "ganglionic system" of branches. They are (1) the lenticular; (2) the lenticulo-striate; (3) the lenticulo-optic. The lenticulo-striate arteries are the most frequently ruptured in cerebral hæmorrhage.

Cerebral Veins.—The veins do not accompany the arteries, but open into the various sinuses in the dura mater. They are arranged in two sets—

1. *The Superficial set*, which open into the superior longitudinal, the lateral, and cavernous sinuses.
2. *The Deep set*, which gather the blood from the *interior* of the brain, and empty into the straight sinus.

The special characters of the cerebral circulation are—

1. The free anastomosis at the circle of Willis, which provides a ready supply of blood from other vessels in case of the sudden blocking of any of the more direct channels.
2. The tortuous course through bony canals of the arteries as they enter the skull, thus mitigating the force of the heart's beat.
3. Their ramifications in the pia mater before entering the substance of the brain.
4. The thinness of the arterial walls, and the smallness of the capillaries.
5. The existence of venous sinuses which are without valves, and which do not run with the arteries; the larger arteries, in fact, having no companion veins. (*Whitaker's Anatomy of the Brain and Spinal Cord.*)

SUMMARY OF THE FUNCTIONS OF THE BRAIN.

It has long been known that the cerebrum contains the highest nerve centres—viz., those centres whose activity is associated with volition, intelligence, thinking, consciousness,

and analysis of sensation, etc.; but it was not proved until 1882 that the cortex is sensible to *direct* excitation. Hitzig and Fritsch in Germany; Ferrier, Horsley, and others in England, have not only proved that the *cortex* itself is sensible to irritation, but, in addition, they have shown that the brain does not act as a whole in all its various functions; indeed, on the contrary, certain parts have special duties allotted to them; in other words, *Stimulation of certain areas causes definite and particular movements, sensations, etc.*

The Localisation of such areas is briefly—

1. *The Frontal lobes*, which are concerned in the higher psychical functions.
2. *The Rolandic area*, associated with motor functions.
3. *The Occipital lobes*, which have to do principally with sensation.

TABULATED, we have the following centres:—

1. The motor centres for the *legs, arms, face, lips, and tongue* (in order from above downwards), in the region of the ascending frontal and ascending parietal convolutions.
2. The centre for *speech* is in the *left* inferior frontal convolutions (Broca's convolution). (If the person be *left* handed, the speech centre is in the *right* convolution.)

Sensory Centres.

3. The centres for *sight* are in the occipital lobe and angular gyrus (see Optic Nerve).
4. The centres for *smell* and *taste* are in the hippocampal region in the temporal lobes.
5. The centres for *hearing* are in the superior temporo-sphenoidal lobes.

The results of stimulation of these centres are seen on the *opposite* side of the body, though sometimes they are bilateral (especially as regards movements of the eyes, trunk, and mouth).

In the case of speech, not only does the cortical centre of one side govern the intricate movements of the mouth and

throat, but the right centre may actually lose its power or function. Cases have been frequently recorded where, through disease of the *left* frontal lobe, speech *for a time* has been entirely abolished, but, regained in time *through the right centre re-acquiring its function*—in these cases the patients have had to learn language as a child does.

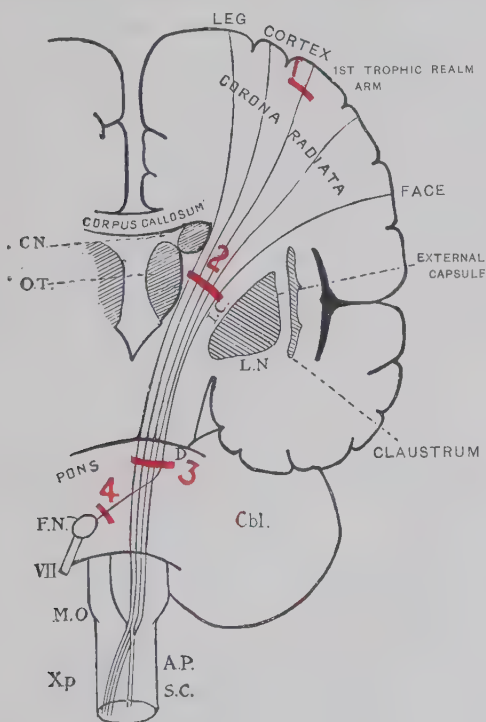
It only remains now for us to consider a few more simple facts; then, I think, brain diseases will be rendered fairly intelligible, even to a beginner.

Note that *irritation* causes increased action; *paralysis* abolishes the functions of the centres. It must also be remembered that though the *cerebral cortex alone* has to do with sensation and voluntary movement, these functions may be abolished by interrupting the afferent and efferent fibres to and from the cortex. The cortex, in fact, is the commander-in-chief; it receives its information by means of inferior officers—basal ganglia, afferent nerves, etc.; and then issues its *orders through a similar efferent mechanism*. The functions of the basal ganglia will now be readily understood. They are subordinate centres conveying and receiving orders from the cerebrum, but they are also able to act, as it were, on their own responsibility as regards certain complex reflexes and co-ordination of movements; thus, the optic thalami contain a large number of sensory fibres passing to the brain, and also a number of *filaments in connection with vision*. Lesions of the optic thalamus or of the corpora striata do not produce *entire* loss of sensation, but only in *proportion to the number of sensory fibres interrupted en route to the brain*. Indeed, when a lesion in this neighbourhood is followed by loss of sensation, it is often partly *due to implication of the posterior limb of the internal capsule*. Hemiplegia, hemianæsthesia, and hemianopia are, however, the symptoms usually described in connection with lesions of the optic thalamus.

Summary of the effect of lesions (destructive and irritative) from the cortex to the spinal cord:—

The Cerebral Cortex.—*Destructive lesions* produce paralysis of the opposite muscles of the body; as the motor fibres are

cut off from the first trophic realms secondary degeneration occurs, causing the paralysis to be of a *spastic nature*. Note that the fibres are spread out in a fan-like manner, so, conse-



DIAGRAMMATIC DRAWING intended to show the effect of lesions interrupting the motor fibres at various levels. Observe a lesion at 1 involves but few fibres ; at 2, a much larger number. Note the relations of the facial nerve at 3 and 4. (See Hemiplegia and Bell's Paralysis.)

quently, the lesion may only affect a few fibres going to one or more groups of muscles.

Irritative lesions are described under Jacksonian epilepsy.

Centrum Ovale.—A lesion will involve a larger number of fibres than in the cortex.

Internal Capsule. --The motor and sensory fibres here converge to the *posterior limb*. A lesion usually produces *typical hemiplegia* (which see).

Crura Cerebri.—A lesion produces a similar hemiplegia, but owing to its relations with the *third nerve*, the *ocular* muscles may be paralysed on the *same side*, *i.e.*, on the same side as the lesion.

Pons.—Note that here the sixth and seventh nerves may be paralysed in the resulting hemiplegia. Remember the crossed nature of the paralysis (see seventh nerve).

Clinical considerations.

Lesions of the cranial contents cause either *irritation* or *paralysis* of the nervous apparatus, motor, sensory, or reflex. Irritation of motor structures is shown by *muscular twitchings* or *spasms*; irritation of sensory parts causes *pain* and *hyper-æsthesia*; irritation of reflex nerve centres leads to *increased reflex action*.

Motor paralysis is estimated by noticing the position of the limbs, the absence of all resistance to passive movements, and stertorous breathing, or flapping of the lips and cheeks with respiration.

Sensory paralysis is recognised by the insensibility of the patient to all external impressions, such as sound, light, pinching, pricking.

Reflex palsy is specially indicated by a fixed condition of the pupils, and the failure of contraction of the orbicularis palpebrarum when the conjunctiva is stimulated.

DIFFUSE IRRITATION.	COMPRESSION.	CONCUSSION.
1. Increased insensibility; patient shows great irritability of temper.	1. Total insensibility.	1. Insensibility, from which patient can usually be partly aroused.
2. Respiration may be quick.	2. Respiration stertorous, slow, and puffing.	2. Respiration feeble, like that of a person in a faint condition.
3. Pulse quick.	3. Pulse full, slow, laboured.	3. Pulse weak, irregular, and often frequent.
4. Pupils contracted.	4. Pupils widely dilated, or sometimes one dilated and the other normal or contracted.	4. Pupils variable, but usually sensitive to light.
5. Reflexes exaggerated.	5. Sphincters may be paralysed, but bowels are torpid.	5. Bowels relaxed, but sphincters not paralysed.
6. Increased irritability of organic reflexes.	6. Bladder paralysed. Consequent retention of urine.	6. Bladder can expel water.
7. Comes on some time after injury, or insidiously from disease.	7. Does not usually appear at moment of injury, but often preceded by signs of irritation.	7. Comes on instantaneously and passes off gradually after injury.
8. Vomiting common.	8. Vomiting may occur, —not usually.	8. Vomiting as recovery is taking place.

We have thus seen that the brain is the initiator or starter of impulses on the one hand, and exercises a controlling or inhibitive power on the other hand. In other words—on the normal exercise of brain function depends the healthy activity of every organ and structure in the body; but the carrying out of this function *is dependent on a perfect maintenance of the anatomical and physiological relations of the component parts.* Lastly, remember that just as the commander-in-chief of an army may be temporarily absent without any great disadvantage, so is it possible for life to be maintained, and, indeed, a fair standard of health to persist for a time, after the higher centres have been destroyed or abolished by disease. Such a condition, however, can only last for a limited period, for the inhibitive power being removed, the reflexes run riot, harmony is replaced by anarchy, premature decay supervenes, and often speedy death terminates a miserable existence. Paralysis of the insane furnishes an example of this.

MENINGITIS.

Inflammation of the meninges of the brain is of two types, *i.e.*—

1. The *simple or traumatic form*, attacking most frequently the convexity of the brain.

2. The *tubercular form*, which principally affects the base of the brain, and is frequently part of a *general* tuberculosis, and always secondary to tuberculosis elsewhere.

THE SIMPLE FORM.

Causes.—Injuries of all kinds to the skull, extension of inflammation from other parts—*i.e.*, disease of the middle ear, irritation from tumours, minute hæmorrhages, etc., or as a complication in fevers, syphilis, etc. The inflammation may be most marked in one membrane, and thus, when the dura mater is principally involved, it is called pachymeningitis; when the arachnoid and pia mater are most affected, it is termed leptomeningitis. Such distinction is of doubtful value in the acute form, though it is well to remember such forms in the chronic variety. Bear in mind also that the dura mater acts as the periosteum to the inner table, so therefore it is easy to see leptomeningitis is most common when the cause is from within the cranium; pachymeningitis when the causes are from without.

Pachymeningitis hæmorrhagica occurs in elderly people with degenerated arteries, especially in general paralytics and chronic alcoholics. On the inner surface of the dura is found a thin membrane, into which small hæmorrhages may occur, or larger hæmorrhages producing a laminated clot. Symptoms are variable. They commence with convulsions deepening into coma. Recovery usually follows, although hemiparesis may persist for some time. There may be other symptoms of cerebral compression. *Fresh attacks occur with each fresh hæmorrhage*, the mental state deteriorates, and dementia develops. Death may occur in an apoplectic seizure. *Treatment* as for cerebral hæmorrhage.

Leptomeningitis :—

Symptoms.—They are divided into three stages.—

1. *Premonitory.*—Headache, more or less constant ; vomiting and restlessness.

2. *Stage of Irritation* is marked by febrile symptoms, great intolerance of light, contracted pupils, exaggerated reflexes, rigidity of the limbs, and convulsions. The patient lies curled up in a characteristic manner, with the head markedly retracted. The pulse, which is at first rapid, becomes slow and *irregular*. Headache is very violent.

3. *Stage of Compression.*—Temperature tends to fall, irritability is replaced by stupor, and convulsions by paralysis. Pupils now become uneven or dilated. Optic neuritis may develop, but frequently there is blindness without marked change in the fundus. The peculiar cephalic cry is a constant symptom. Cheyne-Stokes breathing, involuntary escape of fæces and urine, and stertorous breathing, usher in death, which often takes place very soon after the onset of grave symptoms.

Pathology.—

1. Hyperæmia of the meninges, which become swollen and injected.

2. Exudation of lymph.

3. Effusion, which rapidly becomes purulent.

THE TUBERCULAR FORM.

(“ACUTE HYDROCEPHALUS.”)

The tubercular form of meningitis occurs most frequently in children, but is not uncommon in adults.

The symptoms are also divided into three stages—

1. *Prodromal.*—The child usually shows more or less definite symptoms of the tubercular diathesis, such as emaciation, want of appetite, constipation, alternating with diarrhœa ; irritability of temper, and headache, perhaps form the more common features, until *definite* symptoms of the meningeal affection arise. These symptoms may last a few weeks or months.

2. *Irritative Stage*.—The symptoms are similar to those described under the simple variety, but the head is usually more retracted and neck more rigid; abdomen is hollowed out or boat-shaped, temperature oscillates; internal strabismus may be present, and there is often marked vasomotor paralysis (*tache cérébrale*). Vomiting is very constantly present. It may or may not be related to food. This stage continues for a week or so.

3. *Compression Stage*.—The symptoms that accompany coma develop, and death may take place in from ten days to six weeks from the onset of acute symptoms.

Pathology.—The inflammatory conditions associated with the presence of tubercle, viz.—

1. Invasion of the bacilli into the meninges (pia mater principally), and formation of tubercles.

2. Irritation set up by the tubercles. The tubercles may then go through the stages of (1) softening, (2) bacillary liquefaction (formation of a greenish pus), or (1) fatty degeneration, (2) caseation, with very little lymph, (3) calcification (rare).

The down-grade process commences in the perivascular tissue of the Sylvian and other arteries at the base of the brain, hence the *ventricles are often distended*, and the brain substance flattened up against the skull. Hydrocephalus is thus produced by obstruction of the veins of Galen.

In the adult delirium takes the place of convulsions. The course of the disease is more rapid than in children, as the skull cannot expand, and hence intracranial pressure develops more quickly.

Treatment.—

Simple Meningitis.—Remove any obvious cause; darken the room; shave the head and apply ice; administer a calomel purge, bromides, hyoscyamus, or phenacetin to relieve the headache; and iodide of potassium, or mercury by inunction. Support the patient with fluid nourishment, and prevent bed-sores and over-distension of bladder during the later stages.

Tubercular Meningitis.—This form needs no different treatment after symptoms are developed, but during the prodromal stage everything should be done to combat the tubercular condition by careful hygiene, diet, etc., by the administration of cod-liver oil, fresh air, etc.

DIAGNOSTIC TABLE.

SIMPLE.	TUBERCULAR.
<i>Age.</i> —Any age.	<i>Age.</i> —Young children and young adults.
<i>Cause.</i> — Injury or local causes, fevers, etc.	<i>Cause.</i> — No local cause, but symptoms of tubercle elsewhere.
<i>Course.</i> — Short.	<i>Course.</i> — Longer than simple, especially the prodromal stage.
<i>Convulsions.</i> — They may be present.	<i>Convulsions.</i> — Common, even during the compression stage, often precede death.
<i>Abdomen.</i> —Nothing marked.	<i>Abdomen.</i> —Markedly retracted.
<i>Pathology.</i> — 1. That of simple or suppurative inflammation. 2. Attacks convexity of brain. 3. Ventricles not distended.	<i>Pathology.</i> — That which is associated with the presence of tubercle, and formation of peculiar greenish pus. 2. Attacks the base of brain. 3. Ventricles are distended, and may cause hydrocephalus.
<i>Prognosis.</i> — Almost hopeless.	<i>Prognosis.</i> — Depends on cause and extent.

HYDROCEPHALUS.

Acute hydrocephalus has already been described under tubercular meningitis. Under the term *chronic* hydrocephalus is meant that form arising from—

1. Congenital malformations.
2. Serous effusion into the ventricles during foetal life or early infancy.
3. Obstruction to the return of blood from the ventricles, as by the pressure of a tumour.

Morbid Anatomy.—

Skull.—Sutures fail to unite, and the skull as a whole does not ossify as in health. A characteristic deformity thus develops—viz., overhanging brow, great increase of the circumference of the cranium *and its disproportion to the size of the face*, open fontanelles. The circumference of the head may reach thirty inches or even more.

Ventricles.—They are distended—one, two, or more ventricles may be affected. The lining is granular, and the ependyma thickened. Fluid contains albumen, chloride of sodium, traces of urea, cholesterine, flakes of lymph, etc. The quantity varies—may reach fifteen pints or more.

Brain Substance.—Is much compressed, convolutions flattened out, and cortex much thinner.

Cranial Nerves.—Certain nerves may at first be inflamed, and subsequently atrophy, especially the *optic* nerves.

Symptoms.—The size and shape of the head sufficiently indicate the nature of the disease, even at an early period; later the symptoms that develop are—

1. Arrest of development generally.
2. Impaired digestive functions—distended abdomen, etc.
3. Walking power is slowly gained, or not at all.
4. Mental deficiency, sometimes complete idiocy.
5. Convulsions.
6. Condition of apathy, coma, and death, usually in from five to seven years. Cases have been known to attain the age of thirty years.

Treatment.—Very unsatisfactory, if not hopeless.—

1. Pressure by strips of plaster or elastic bands.
2. Puncturing at various intervals, and drawing off the fluid. The ventricles may be tapped, and direct drainage established, or the fluid may be more slowly removed by lumbar puncture (see p. 43).

SINUS THROMBOSIS.

This is the formation of blood-clot in one or other of the intracranial sinuses. It may be simple or infective. The *simple form* occurs in ill-nourished children, and in the debilitating diseases of adults (phthisis, cancer, fevers, anæmia). The longitudinal sinus is most often affected. Its walls are not inflamed, but the lumen is filled with adherent clot, which tends to become organised and absorbed. *Infective thrombosis* follows middle ear disease, caries of other parts of the skull or of the teeth, retropharyngeal abscess, erysipelas of the scalp, etc. The lateral sinus is oftenest affected. Its walls are inflamed; the clot breaks down and becomes purulent, and meningitis or cerebral abscess may occur.

Symptoms may be absent in simple thrombosis. There are generally headache, vomiting, convulsions, or delirium, and drowsiness deepening into coma. There may be epistaxis from engorgement of the nasal veins. In children the fontanelle may be prominent. In thrombosis of the cavernous sinus the orbital veins are engorged and the eyelids œdematous; the fundus shows retinal hæmorrhages and engorged veins; and there may be paralysis of ocular muscles. In thrombosis of the lateral sinus the mastoid region is œdematous and tender; the thrombosis extends to the jugular vein, which can be felt in the neck as a hard, tender cord; there is local meningitis, and possibly optic neuritis. There is a purulent discharge from the ear.

In infective thrombosis symptoms of general sepsis are superadded,—vomiting, rigors, profuse sweating, remittent fever, and rapid irregular pulse. Septic pneumonia, due to pulmonary infarction, may follow, or an abdominal type of infection,—dry tongue, vomiting, diarrhœa, and meteorism, ending in a typhoid state. Meningeal symptoms are also present, and death is preceded by coma.

Treatment.—In the simple form treat the constitutional state, and favour free circulation through the brain, particularly

avoiding constriction of the neck by clothing. In the infective form the treatment is surgical.

ENCEPHALITIS.

Encephalitis is an inflammation of the substance of the brain, which may be either hæmorrhagic or suppurative, acute or chronic. The acute form may be focal or diffuse, in the chronic form there are scattered inflammatory patches. The hæmorrhagic form is associated with infective diseases such as enteric, influenza, diphtheria, syphilis, and with the exanthemata. The suppurative form follows suppuration of the accessory cranial cavities,—ear, orbit, nose.

Acute Hæmorrhagic Encephalitis (focal) attacks the grey and white, but especially the grey, matter of the mid and hind brain and of the cortex. The nuclei of cranial nerves are thus involved. The condition is analogous to acute anterior poliomyelitis (*q.v.*). The affected parts are found to be softened, and show engorgement and thrombosis of vessels, hæmorrhages, and proliferation of leucocytes, the nervous elements being destroyed.

Symptoms.—Sudden onset ; headache, vomiting, convulsions, fever, drowsiness leading to coma and death, or partial recovery. Optic neuritis is sometimes seen. Local symptoms are sudden ophthalmoplegia (*polioencephalitis superior*), or sudden bulbar paralysis (*polioencephalitis inferior*). The nuclei of the fifth and seventh may also suffer.

Acute Diffuse Encephalitis is rare. It is oftenest seen in children, when it may follow infections or be caused by traumatisms. In the adult it is associated with syphilis. Large areas of brain on one or both sides are involved in the inflammatory process, which leads to softening. Later the affected portions may become atrophied or cystic (porencephaly). The disease may be fatal, but more often ends in recovery, leaving behind it, however, paralysis of a cerebral type—infantile hemiplegia or diplegia—and in some cases epilepsy.

SUPPURATIVE ENCEPHALITIS— ABSCESS OF THE BRAIN.

The causes of cerebral abscess are to be found chiefly in disease of the accessory cavities, especially the *middle ear*, but disease of the cranial bones, fracture or gun-shot wound of the skull, may give rise to it, and also infection from a distance (metastatic abscesses), as in septic pneumonia or peritonitis, osteomyelitis, or pyæmia. The temporo-sphenoidal lobe is most often affected, and next the cerebellum. Any part of the brain may suffer, according to the seat of the causative disease.

Morbid Anatomy.—The abscess is usually situated in the white matter, and may be separated from the surface by healthy brain tissue, the infection having been carried by the venous sinuses and perivascular lymphatics. Usually there is a localised meningitis, the dura adhering to the brain. If the abscess is acute, the pus is not encapsulated, but surrounded by broken-down nervous tissue; if chronic, there is a definite capsule.

Symptoms of Acute Abscess.—Pain in the ear, radiating over the side of the head; cessation of aural discharge; rigors, and slight fever; vomiting. After a few days, lassitude, drowsiness, inability to fix the attention or to answer questions; temperature normal or subnormal; pulse slow and full; respirations slow, tending to Cheyne-Stokes type; optic neuritis in the later stages; sometimes cerebral palsies; convulsions *uncommon*, unless the Rolandic area is involved. Ultimately the lethargy deepens into coma and death, which may be sudden from rupture into the lateral ventricles. *Localising symptoms*:—

1. *Temporal lobe.* Paralysis of the third nerve on the side of the ear disease. Hemiplegia on the opposite side, most marked in the face, least in the leg. Word-deafness when the left first temporal convolution is affected.
2. *Cerebellum.* Giddiness and staggering gait, tendency to fall to the side; retraction of head and stiffness of muscles of neck; nystagmus, deviation of the eyes

away from the side of the abscess ; sometimes hemiparesis on the same side as the abscess with variable intensity of the knee-jerk, which is sometimes exaggerated.

3. *Frontal lobe.* Localising signs may be absent, or there may be paralysis of the limbs (arm especially) and face on the opposite side, with aphasia, if the abscess is on the left side.

Remember that abscess may be entirely latent, or lead only to a more or less transitory irritability, inertia, and headache. After these have passed off, injury to the head or relapse of a previous ear trouble may, even years afterwards, light up acute symptoms and lead to death.

Treatment.—Attend at once to all chronic suppurative conditions of the ear, nose, or frontal sinuses (prophylaxis), and employ strict antiseptic precautions in dealing with erysipelas or scalp wounds. When abscess is actually present, immediate operation is the only treatment, even in the absence of localising symptoms. A clue to the site of the abscess will be found in the seat of the primary disease.

CEREBRAL HÆMORRHAGE.

Ætiology.—It occurs most frequently in men between the ages of forty and sixty, but may occur at any age. Amongst the more important predisposing causes are—

1. A certain type of build. It is frequent in stout, plethoric men, with short, thick necks.

2. Certain occupations—viz. (1) butchers, publicans, by producing arterial changes through excessive consumption of nitrogenous food and alcohol ; (2) carters, hammermen, etc., by producing vascular strain.

3. Certain blood diseases—leucocythæmia, pernicious anæmia, scurvy.

4. Degenerations due to chronic renal disease, the rupture being aided by the forcible pulsations due to cardiac hyper-

trophy. Syphilitic degenerations may lead to hæmorrhage, but more frequently cause thrombosis.

5. Removal of the natural vascular *support*, as in cerebral softening.

6. Injuries from without the cranium, etc.

The most important immediate factors, however, are—

1. Disease of the vascular walls.

- (1) Miliary and other aneurismal dilatations.
- (2) Atheroma.
- (3) Arterio-sclerosis.
- (4) Degeneration due to chronic alcoholism or gout.

2. Vascular strain, as from exercise, or causes such as straining at stool, or violent coughing.

Site of the Hæmorrhage.—The most common form of cerebral hæmorrhage is *into the substance* of the brain, and is usually due to rupture of the lenticulo-striate and lenticulo-optic branches of the middle cerebral artery, which supply (as we have seen) the basal ganglia. These arteries are the most frequent seats of miliary aneurism, and the reason why they are so frequently ruptured, is probably that they are the most direct branches of the middle cerebral, which is in turn the most *direct* branch of the internal carotid. Meningeal hæmorrhage may also take place, as in depressed fracture of the skull or pachymeningitis hæmorrhagica.

Anatomical Changes.—If the hæmorrhage be severe it tears up the brain tissue, or it may destroy the basal ganglia, and bursting into the lateral ventricles distend them, and flow through the aqueduct of Sylvius into the fourth ventricle. The hæmorrhage, however, may be small. The subsequent changes in the effused blood are of the utmost importance, viz.—

1. *Changes in the clot*—

- (1) Retraction, and exudation of serum (which is partially absorbed).
- (2) Discoloration of the clot through infiltration of

leucocytes, and partial breaking-down of red corpuscles.

- (3) Formation of hæmatoidin crystals. Hæmatoidin is a form of reduced hæmatin which contains no *iron*.
- (4) Formation of a serous cyst. Occasionally there may be complete absorption, when only a fibrous cicatrix is left.

2. *Changes in the tissues around—*

- (1) Irritation causing hyperæmia.
- (2) Diapedesis of leucocytes, and proliferation of the tissue cells.
- (3) Increased fibrous formation, forming a capsule around the clot.

3. *Changes in the fibres* interrupted by the hæmorrhage, and thus cut off from the first trophic realm (cortex). Secondary descending degeneration occurs of the motor tracts on the affected side, and after the decussation in the medulla, on the opposite side (crossed pyramidal tract).

Summary.—After cerebral hæmorrhage, if the patient survives the primary shock (the apoplectic fit), he has to run the gauntlet of—

1. Reaction. (Inflammation of brain substance, softening, and abscess formation *may* occur.)
2. Resulting motor paralysis (hemiplegia).
3. Secondary degeneration of motor fibres, and possible involvement of important nerve nuclei.

Symptoms.—There may be premonitory warnings of the attack, such as giddiness, headache, etc. The attack may occur when the patient is resting or asleep, but much more frequently is it directly attributable to an overloaded stomach, a severe strain, coughing, or the exertion in running to catch a train. Apoplexy is not synonymous with cerebral hæmorrhage; still, the condition which supervenes on such a lesion is generally summed up in the description of an—

Apoplectic Fit.—The patient is suddenly seized with severe

pain in the head, feels faint, giddy, and quickly falls into a state of collapse, which in a short time passes into a comatose condition. The face is flushed, the pulse full and tense, breathing stertorous, and cheeks puffed out. The snoring noise is due to the paralysed tongue and palate falling back, and impeding the entrance of air. The pupils are dilated or irregular. The limbs at first are flaccid, but soon become somewhat rigid (so-called *early rigidity*). The skin reflexes are lost, but the deep reflexes are increased after a few days. There are inability to swallow, retention or involuntary dribbling of urine, and involuntary evacuations. The head and eyes are usually turned to one side—*i.e.*, patient looks towards the lesion *away* from the paralysed side—(conjugate deviation), owing to the balancing action of the associated muscles being lost from paralysis. When the case is going to end fatally, the coma increases, temperature falls, and Cheyne-Stokes respirations usher in the fatal end. In favourable cases the primary disturbance is not so grave, and the patient may regain consciousness in a few hours, or even after two or three days. “Reaction” then sets in, the temperature rises, as does the pulse, perspiration occurs, and there is restlessness, with excitement amounting sometimes to delirium. Recovery is rarely complete, but usually attended by more or less paralysis or typical hemiplegia.

It is possible even in the comatose condition to make out the existence of paralysis. The cheek is more puffed out on the affected side, the naso-labial groove is obliterated, and the angle of the mouth is lower, while saliva trickles from it. If the limbs upon the two sides be raised alternately and then let fall, those on the paralysed side fall in an absolutely flaccid fashion, while upon the other side some tone is still maintained.

EMBOLISM AND THROMBOSIS.

Instead of a free extravasation of blood we may get blocking of blood-vessels through detached clots (embolism), or a local clotting of blood (thrombosis). The following table shows the more important points in the diagnosis between the two.

DIAGNOSTIC TABLE.

CEREBRAL EMBOLISM.

Causes.—

Most commonly associated with valvular lesions of the heart, aneurisms, or a suppurating thrombus.

Age.—

Chiefly in young adults.

Onset.—

Sudden—no prodromal symptoms.

Convulsions.—

Rare.

Paralysis.—

A sudden hemiplegia of the right side, *with aphasia*. The left middle cerebral artery is most frequently plugged.

Consciousness.—

Not usually entirely abolished.

CEREBRAL THROMBOSIS.

Causes.—

1. Atheroma; and morbid states of the arteries, as seen in chronic Bright's disease, syphilis, pyæmia, etc.

2. Pressure upon the veins or arteries.

Age.—

In the old from vascular degeneration; in the young from syphilis.

Onset.—

Gradual—with prodromal symptoms.

Convulsions.—

Convulsive attacks are not uncommon, as the cortex is frequently affected.

Paralysis.—

Paralysis is more gradual. Aphasia is not so common, as either the right or left side may suffer.

Consciousness.—

Often not lost. Coma may come on gradually, after the hemiplegia.

Lastly, we must remember that hæmorrhage, embolism, or thrombosis may be confined to the small vessels of the cortex alone.

In cortical hæmorrhage—

1. Consciousness is seldom completely lost at the onset of paralysis.
2. The resulting paralysis is of a "monoplegic" type.
3. The paralysis is often transitory.
4. Rigidity of affected muscles comes on earlier.
5. Prognosis is much more favourable.

Anatomical Changes following thrombosis and embolism will vary according to the site and size of blood-vessels affected. Briefly, the ultimate results are degeneration and softening of the parts supplied by the affected artery. Blocking of a terminal artery causes the affected area to look slightly paler at first ; then extravasation from the engorged veins or collateral circulation may take place and cause an infiltration of blood. The resulting softening may be of the red, yellow, or white types, and the clot undergoes the changes before described. Two things the student must remember—

1. If the clot be of a *septic* nature, an abscess is likely to form.

2. If *not* septic, it is surprising how slowly the symptoms dependent on softening develop.

Thrombosis may follow upon embolism, a small branch being first blocked, and the thrombus extending backwards till it blocks a large branch or the main trunk of the middle cerebral artery.

Blocking of particular arteries.

1. *Vertebral*.—Rare, left more common than right. The symptoms are those of acute bulbar paralysis.

2. *Basilar*.—Bilateral paralysis with *rise of temperature*.

3. *Posterior Cerebral*.—Affections of vision (hemianopia) and hemianæsthesia (posterior portion of internal capsule).

4. *Middle Cerebral*.—Hemiplegia with aphasia.

5. *Anterior Cerebral*.—Grave interference with the higher intellectual faculties.

Treatment of Cerebral Hæmorrhage.—Place the head high, and put ice to head, and counter-irritants (mustard and hot-water cloths) to feet ; administer an enema, calomel grains v. on the tongue, or a minim of croton oil. Venesection should be performed if the pulse is full and of high tension ; if, on the other hand, the pulse be weak, or we are fairly certain the symptoms are due to thrombosis or embolism, it is best not to resort to bleeding. If the case be syphilitic, begin inunction of mercury at once, and later iodides. Relieve

stertor by turning the patient on his side. Draw off the urine by catheter. Feed by the rectum if the coma is prolonged. The resulting paralysis must be treated on the general principles detailed in the chapter on Treatment of Chronic Diseases of the Cord.

HEMIPLEGIA.

By hemiplegia is meant paralysis of the face, arm, and leg on one side of the body, resulting from a lesion of the opposite cerebral hemisphere. The movements of the muscles of the trunk (especially the respiratory muscles), and those of the vocal cords, are not usually affected, inasmuch as they are bilaterally innervated, and the destruction of one centre means that the opposite centre assumes its functions.

Causes.—Hemiplegia is most commonly the result of embolism, thrombosis, or a free hæmorrhage. It may be caused by depressed fractures involving the Rolandic area. It may be due to a tumour involving the motor fibres anywhere between the cortex and the medulla, in which case it develops gradually. In hæmorrhage the motor fibres are torn; in embolism or thrombosis there is softening of the cerebral tissues.

Symptoms.—When the hemiplegia is not preceded by an apoplectic fit, the patient often first becomes aware of anything wrong by finding on awakening from sleep

1. Loss of power on one side, or
2. Difficulty in speaking, or even loss of speech (dysarthria or anarthria). This is to be distinguished from *aphasia*, which may be present in right-sided hemiplegia if Broca's convolution on the left side is implicated in the lesion, but is not present in left-sided hemiplegia.

In "ingravescent hemiplegia" (tumour), unconsciousness may develop slowly and pass into complete coma. In the ordinary forms coma may be present at the outset, and pass away, or there may be only a feeling of cloudiness or bewilderment, which may pass off or deepen into coma.

The arm is usually paralysed to a greater degree than the

leg, and the face least. The face usually recovers before the leg, and the leg before the arm, while coarse movements of large joints are re-established before the highly specialised movements of small joints. Improvement is therefore least in the hand and fingers. Sensation is rarely affected to a marked extent. The paralysis of the face is most notable in its lower segment, thus differing from Bell's paralysis (see Diseases of Cranial Nerves). The tongue is not put out readily, and deviates towards the *paralysed* side. Whistling is impossible, and food accumulates between the gums and cheek on the paralysed side. The muscles of mastication are unaffected. In the limbs the muscles do not waste, and they respond normally to electricity. They are at first flaccid, but may become rigid a few hours after the initial lesion has occurred ("early rigidity"). This may pass off, or persist till "late rigidity" appears some weeks afterwards. As time wears on, certain important symptoms appear, as a result of the secondary degeneration of the motor fibres cut off from their trophic centres. The limbs, before flaccid, now become rigid, and this late rigidity is always most marked in the arm, which is adducted, flexed at the elbow, and resists extension. Frequently the wrist and fingers are also flexed. The tendon reflexes are exaggerated, and ankle clonus is often obtained. The plantar reflex is of the extensor type (Babinski's sign). As the patient walks, the body is inclined to the normal side, and, since the foot is dropped, he either sweeps the affected leg forward in a circular arc, or flexes the knee unduly, to clear the toes from the ground. After recovery from the initial coma, the sphincters are unaffected, and control over the urine and faeces is regained.

Later changes that may occur are—tremors of the affected limbs, inability to maintain one position of the fingers and toes (athetosis), and post-hemiplegic chorea. The two last are more common after infantile than adult hemiplegia. There may be vasomotor disturbances, arthritic pain and swelling, and sometimes muscular atrophy (chiefly in the hand), from unilateral neuritis.

Alternate or crossed paralysis means paralysis of the limbs on one side, and of a cranial nerve or nerves on the other. It occurs when the lesion is in the crus, the third nerve of the same side being implicated, or in the pons, when the fifth, sixth, or seventh nerves may be affected (see Diseases of Cranial Nerves, and diagram p. 423).

Treatment.—Endeavour to prevent the onset of rigidity and contracture by the prolonged employment of passive movements and of galvanism. These may be begun as early as four weeks after the onset, at first while the patient is in bed. Later “Swedish movements” may be employed. Faradism is to be avoided, and so also is strychnine, from its liability to cause rigidity. Treat any general condition such as Bright’s disease.

CEREBRAL PALSIES OF INFANCY AND CHILDHOOD.

These affections are caused by pathological changes occurring before or during birth, or in the early years of childhood. Maternal disease or injury during pregnancy, injury during birth, and after birth the infectious diseases of childhood, are the chief causes.

Morbid Anatomy.—Only the late changes are well known, as death rarely occurs in the early stages. The probable antecedent conditions are mal-development, meningeal or cerebral hæmorrhage, arterial thrombosis, and encephalitis. It is always the cerebrum that is affected, and lesions may be found on one or both sides. These conditions lead in after years to (1) sclerosis of the convolutions (*microgyria*), in which the convolutions are small and the cortical cells atrophied; (2) cavity formation (*porencephaly*), which follows upon vascular thrombosis or encephalitis.

Symptoms.—*Hemiplegia*. In this type are found paresis, rigidity, contracture, and often tremors of one side of the body. There may be convulsions at the onset. The affected side is

ill developed, and shows marked atrophy. There may be athetosis, or choreiform movements. This form arises within the first two or three years after birth, and follows infectious diseases.

Diplegia and paraplegia constitute the more usual "birth palsies." There is often a history of difficult labour. There are paresis, rigidity, and contracture of the legs, with adductor spasm; there may or may not be paresis of the arms, which show athetoid or choreiform movements. Sometimes the lower segment of the face is implicated. The tendon reflexes are exaggerated. The gait is spastic, and there may be cross-legged progression from the adductor spasm. Deformities such as club-foot are frequent.

In all these forms some degree of mental impairment, which may even amount to idiocy, is commonly present. Epilepsy may also follow, particularly in the hemiplegic form, the paretic side being specially affected. This may not occur till many years have passed.

These palsies are not dangerous to life, but seriously affect growth and mental development. They are to be *treated* on general principles. The child should be educated separately, and much attention should be given to physical training. Deformities may require surgical treatment, and epilepsy prolonged treatment by bromides.

CEREBRAL SYPHILIS.

Syphilis affecting the brain may give rise to vascular lesions (obliterative endarteritis), and gummata, or to degenerative diseases. The vascular lesions may occur early—and sometimes quite early—the degenerative conditions (see General Paralysis) much later. The severity of the attack of syphilis has little influence on the development of nervous lesions, and they may follow even prolonged and careful treatment.

Morbid Anatomy.—Obliterative endarteritis causes a uniform narrowing of the lumen of the cerebral arteries and

arterioles—the basilar and vertebrals, and the middle cerebral and its branches being chiefly affected. The arteries of the basal ganglia do not suffer so often. The lumen of the affected artery is ultimately obliterated by thrombosis. There is usually periarteritis, with formation of gummatous nodules. Complete occlusion of course leads to cerebral softening.

Isolated gummata—single or multiple—may be found over the cerebral convexity, at the base, on the cranial nerves, or in the substance of the brain. A diffuse gummatous meningo-encephalitis may also occur, especially at the base.

Symptoms are similar to those produced by other cerebral lesions, but they may be combined in a suggestive way. Signs of double or multiple lesions, a tendency to improvement and relapse, and a tendency to improvement under treatment, are all significant. Headache, usually worse at night, vertigo, insomnia, apathy, epileptiform convulsions or fits, amnesia, mental confusion, are frequent prodromata. From *vascular occlusion* hemiplegia and aphasia may arise. The onset is gradual, generally without unconsciousness, the paralysis is often but not always temporary, and it may be followed by another attack on the same or the opposite side. From *cortical gummata* arise localised convulsions (Jacksonian epilepsy), with only incomplete unconsciousness, and often monoplegia. The paralysis comes on slowly, and is persistent. Optic neuritis sets in early and is very intense. From *gummatous meningitis of the base* arises paralysis of the cranial nerves. All of these may be affected, but most frequently the third or sixth, or both of them. Optic neuritis is exceptional, but bi-temporal hemianopia from gumma in the interpeduncular space may occur. There are usually other signs of intracranial mischief (headache, vertigo, etc.). From *diffuse arterial and meningeal lesions* arises syphilitic dementia, which may be accompanied by local or general convulsions, aphasia, affections of cranial nerves, and more constantly by amnesia, mental confusion, somnolence, and melancholia or sometimes mania.

Prognosis depends upon the stage of the cerebral affection. If vascular occlusion has led to softening, or if fibrous trans-

formation has occurred, there will not be much improvement; while early cases may be immensely improved, or even cured, by energetic treatment.

Treatment.—In cerebral syphilis the more vigorous forms of treatment are chiefly employed. Mercury is of the first importance. It may be given by inunction either of the ordinary Ung. Hydrargyri, or of Ung. Hydrargyri Oleat., which is cleaner and less irritating. Intra-muscular injection of a soluble salt is also strongly advocated. Iodides may be given alone or in combination with mercury. The doses should be large—15 to 30 grains of the iodide of potassium thrice daily. Such doses are less likely than smaller doses to produce iodism. Sulphur baths aid the elimination of mercury from the system, and thus permit the use of larger doses.

TUMOURS OF THE BRAIN.

These are of three kinds—(1) Infective granulomata—tubercle, gumma, actinomyces; (2) neoplasms—sarcoma, glioma, carcinoma, and benign tumours; (3) cysts.

The most important are—

1. *Tubercle.*—The tumours may vary in size from a millet seed to a small orange. Usually they are multiple, and most common in the cerebellum and about the base of the brain. Histological characters are the same as already described under tuberculosis.

2. *Gummata* are very common (see Cerebral Syphilis).

3. *Glioma, or neuro-glioma.*—They are usually dense, firm tumours, but *may be soft and vascular*. Histological characters,—they consist of round or oval nucleated cells, with branched processes, forming a network. *Often enormous, single-nucleated, spindle-shaped cells are present.* They are usually innocent tumours, and may remain for years without creating very marked symptoms.

4. *Sarcoma*—attacks the membranes and pons, but may be found in other parts of the brain. They may be large

tumours, hard, fibrous, and encapsulated, or soft, infiltrating, and sometimes cystic.

5. *Sarco-glioma*—found chiefly in the retina. They differ from simple glioma in a tendency to assume a malignant character.

6. *Carcinoma*—rare—secondary to cancer elsewhere.

7. *Cysts*—may be the result of—

(1) Hæmorrhage.

(2) Congenital defects.

(3) Hydatids.

Symptoms.—They vary according to the site, size, and nature of the tumour. The more constant symptoms are—

1. *Headache*.—May be diffuse, and dull aching in character, but is much more often of an acute stabbing nature, persistent, and localised. There is sometimes tenderness over the seat of the tumour.

2. *Vertigo*.—A most marked symptom, especially when the tumour affects the cerebellar region.

3. *Vomiting*.—Very persistent, and occurs whether food be taken or not. It is most common when the cerebellum is affected.

4. *Double Optic Neuritis*.—Choked disc (is almost pathognomonic). It may be slight in cortical tumours, but is present in 80 per cent of all cases. It is followed, by optic atrophy. Blindness is not necessarily present.

5. *More or Less Mental Disturbance*.—Sometimes absent, but the patient is sometimes, in the early stages, highly emotional, and later, dull and apathetic.

6. *Localising Symptoms*.—These may be absent, but usually there are phenomena either of irritation or destruction of the affected part of the brain, the former leading to convulsions, paræsthesiæ, and subjective sense impressions, the latter to paralysis, anæsthesia, and defect of special sensation.

A. Prefrontal area. Lethargy, somnolence, stupor, dementia; often anosmia on the affected side.

B. Rolandic area. Jacksonian epilepsy followed by monoplegia; increased tendon reflexes on the paralysed side; motor aphasia if Broca's convolution is affected.

C. Angular region. Word-blindness with or without hemianopia and hemianæsthesia.

D. Basal ganglia. Hemiplegia, hemianæsthesia, and hemianopia.

E. Cerebellum. General symptoms severe. Ataxia, with tendency to fall to one side; unilateral affections of cranial nerves (fifth and eighth usually); knee-jerks absent or exaggerated.

Diagnosis.—The differential diagnosis between tumour, abscess, and tuberculous meningitis is given in the following table:—

TUMOUR.	ABSCESS.	MENINGITIS.
History indefinite.	Otorrhœa or other suppurative condition.	Tuberculous history or diathesis.
Onset gradual.	Onset usually abrupt.	Onset rapid.
Optic neuritis usually well marked.	Optic neuritis usually absent or late.	Optic neuritis rare.
Monoplegia, hemiplegia, or localised convulsions, in definite order.	Focal symptoms indicative of cerebellum or temporal lobe.	Irregular palsies and convulsions.
Febrile symptoms absent.	Temperature sometimes subnormal.	Temperature irregular.
Duration months to years; regular course.	Duration variable with latent periods.	Duration of weeks, at times irregular.

[ALDREN TURNER.]

Treatment.—

Medical.—If syphilitic, iodide of potassium in heroic doses; other forms are practically unamenable to any known medicinal treatment. The iodide should nevertheless be given, as it often produces temporary benefit. Tuberculous tumours may be treated by the usual remedies.

Surgical.—Trepine when necessary for the relief of pressure, and remove if possible, but few cases are available for this treatment. Consult a surgical work for details.

Though so many cases cannot be *permanently* benefited by treatment, everything should be done to make the patient's life bearable, by relieving pain, etc., with opiates or by other means.

APHASIA.

Aphasia is a morbid condition consequent on a cortical lesion whereby speech, writing, or reading may become impaired either from (1) an inability to co-ordinate the necessary movements involved in speech, or (2) from a defective interpretation of sounds or visual impressions.

To understand the various forms of aphasia it is necessary first to consider the factors employed in expressing our thoughts. Language is *gradually* learned. For instance, a child has first to learn the movements necessary to articulate the word. Having learned the word, he then associates it with a particular something he has seen or heard. Later, he learns the alphabet—that is, he invests certain letters with definite sounds and meanings, and as his vocabulary increases he can express ideas in speech, and finally spell and write. Thus, intelligent and rational speech, though apparently simple, involves many complex processes. It requires—

1. The aid of memory to *co-ordinate the necessary ideas* formed by incoming impressions derived through hearing, seeing, and in the case of the blind, through the sense of touch.

2. A mechanism by which these ideas can be spoken and written.

In short, speech involves a healthy continuity between—

The chief motor centre in the left inferior frontal lobe and the visual and acoustic centres on the one hand, and—

The chief motor centre and the muscles employed in speech or reading aloud.

There are four centres, two motor and two sensory, concerned in speech and writing. The motor centres are,—for speech, the *glosso-kinæsthetic centre* in the third left frontal convolution (Broca's convolution); for writing, the *cheiro-kinæsthetic centre*, in the posterior part of the second left frontal convolution. The sensory centres are the *auditory word centre*, in the posterior two-thirds of the first temporal convolution, and the *visual word centre*, in the angular gyrus and adjacent part of the supramarginal convolution. The two word centres are connected by commissural fibres, and the auditory word centre is similarly connected with the glosso-kinæsthetic, the visual with the cheiro-kinæsthetic centre. Similar centres exist in the right cerebral cortex, but are in health. entirely subordinate. They have, however, a limited capacity for taking over the functions of the left centres if these are destroyed by disease.

1. Lesions of the glosso-kinæsthetic centre cause *motor aphasia*—the patient is unable to express himself in words, although there is no paralysis of articulation, and he shows that he understands what is said to him.

2. Lesions of the cheiro-kinæsthetic centre cause *agraphia*,—the patient cannot express himself in writing.

3. Lesions of the visual word centre in the occipital lobe cause *word blindness* or *alexia*—*i.e.*, the patient can see print, but cannot read it.

4. Lesions of the auditory word centre in the temporo-sphenoidal lobe cause *word deafness*—the patient hears, but does not understand words.

(3) and (4) are both forms of sensory aphasia, (1) and (2) of motor aphasia. But the terms are loose, for word deafness is usually followed by motor vocal aphasia even if the lesion be limited to the auditory word centre, and word blindness is similarly followed by agraphia. It is impossible here fully to describe the various forms, but the following table indicates the chief differences between motor and sensory aphasia.

MOTOR APHASIA.	SENSORY APHASIA (VERBAL AMNESIA).	
	WORD DEAFNESS.	WORD BLINDNESS.
Patient almost completely loses power of speech. Words like oaths, "yes," or "no," may be retained.	Can still speak, sometimes with little aphasia, but sometimes merely gibberish.	Speech little affected.
Understands what is said to him.	Does not understand what is said.	Understands what is said.
Cannot repeat words.	Cannot repeat words.	Can repeat words.
Recognises written words but cannot write them. Cannot copy print into writing, though he may copy letters (aphasia and agraphia). Rarely can write (aphasia without agraphia).	May be some word blindness and agraphia, or patient may recognise and write words freely.	Cannot recognise written or printed words, or write them (agraphia). May recognise letters, of his own name. If the damage is partial, may write wrong words or in wrong order (<i>paragraphia</i>).
Is aware of his errors—he can recall words but not utter them.	Is unaware of his errors of speech—auditory word memory is destroyed.	Is unaware of his errors in writing—visual word memory is destroyed.
Mental impairment is but slight.	Mental impairment is marked.	Mental impairment is slight.

DISSEMINATED SCLEROSIS.

Disseminated sclerosis, or the so-called "insular" paralysis, is a disease characterised pathologically by scattered patches of sclerosis throughout the central nervous system. The condition may be most marked in the brain, or in the spinal cord; but the *cerebro-spinal* variety is much more common.

Ætiology.—Occurs in youth or middle age; both sexes are liable to the affection. Exposure to cold, injuries, and mental worry are possible predisposing causes. The real cause is not known, although it may be a toxic agent.

Pathology.—Patches of sclerosis, sharply defined, and varying in colour from pink to ashy grey, some hard and leathery, others soft, are found scattered throughout the white matter of the cord and the substance of the medulla and cerebrum, and sometimes the cranial nerves. Their presence causes pressure on the nerve-cells and fibres. The former

atrophy, the latter do so partially, losing the white substance of Schwann, but retaining the axis-cylinder. Hence there are no extensive ascending or descending degenerations.

Symptoms.—With such widespread pathological changes it is practically impossible to say what symptoms may or may not be present in a given case, as the symptoms will of course depend on the site and extent of the sclerosed areas. Taking a typical case, however, we usually find the following symptoms more or less marked :—

1. Impaired Speech.—The person has a slow, monotonous way of pronouncing every syllable distinct, like the staccato delivery of music.

2. A peculiar tremor of muscles, which becomes very marked when voluntary movements are attempted (“intention tremor”); the movements of the hands are specially embarrassed by the tremor, and directed with difficulty.

3. Nystagmus, worse on trying to fix the eyes.

4. Slight impairment of sensibility, but no lightning pains or girdle sensation.

5. Impaired intellectual functions, as loss of memory, etc.

6. More or less paresis, at first of the lower limbs, with spasm.

7. Rigidity of the lower limbs in the position of extension follows later.

8. Exaggeration of the tendon reflexes.

9. Peculiar Gait.—There is marked spastic weakness of not only the legs, but *arms, trunk, and head*. When walking, the patient shoots suddenly forward or to one side, and may fall, or bruise himself by bumping against various obstacles. There is often a mixture of ataxia with paraplegia.

10. The sphincters may or may not be affected.

11. Occurrence of peculiar apoplectiform or epileptiform attacks, from which the patient recovers in one or two days.

The course of the disease is protracted (five to ten years). In the late stages dementia is common. Death may be due to an apoplectiform attack, or to extension of the changes to the grey matter, leading to paralysis of the bladder, bed-sore, etc.

Diagnosis.—The characteristic gait and speech usually render the diagnosis easy, but remember that the dissemination of the lesion might be due to an extension of a hitherto *local* lesion. The diagnosis between disseminated sclerosis and general paralysis sometimes offers difficulties, and depends chiefly on the recognition of the peculiar mental symptoms in the early stages of the latter disease.

The *treatment* is similar to that of chronic diseases of the spinal cord.

GENERAL PARALYSIS OF THE INSANE.

(PARALYTIC DEMENTIA.)

A condition marked by progressive derangement of the mind accompanied by progressive paresis and alteration of the deep reflexes.

Ætiology.—The disease seldom occurs before twenty years of age—most frequently in the thirties. The average age of death is forty. It affects all classes, but chiefly those living in large towns. Men are much oftener attacked than women. The most important cause is syphilis. Mental worry or overwork, alcoholism, and injury to the head are predisposing causes.

Pathology.—The post-mortem appearances vary greatly. In some cases little anatomical change has been discovered; but usually the following conditions exist:—

1. Thickening of the inner table of the skull, which is marked by Pacchionian bodies.
2. Thickening of the membranes, with hæmorrhages between the brain and dura mater.
3. Degeneration of cerebral blood-vessels.

4. Patches of sclerosis throughout the white matter of the cord and brain.
5. Atrophy of the frontal and central lobes.
6. Distension of the ventricles by fluid.

Microscopically, the following changes are found: Thickening of the pia arachnoid, overgrowth of the neuroglia, partial or complete destruction of the nerve-cells. The association fibres are absent or greatly diminished. It is "a disease of impaired metabolism of the neurons" (MOTT).

Symptoms.—It is impossible to give a typical clinical description, as individual cases vary so much. Perhaps it is best to describe three stages:—

First Period marked by changed mental conditions such as—

1. Irritability of temper, jealousy, hallucinations.
2. Loss of memory.
3. Defective speech—patient is unable to repeat similar or many-syllabled words.
4. Exaltation of ideas, and loss of judgment. Patient may fancy he is rich, or may buy a stud of horses, order a banquet, etc.
5. Perversion of the moral faculties.

Second or Epileptiform Period.

1. Patient has a series of convulsions which render his mental condition worse than before.
2. Symptoms of paralysis appear; first, in the execution of fine movements, such as writing, etc.; later, the paralysis involves the face and tongue, or paresis of the limbs and common sensibility develops.
3. Speech is markedly altered, becoming jerky and irregular.
4. The pupils are unequal and sometimes irregular. The Argyll-Robertson pupil may be present.

Third or Paralytic Stage.

The mind finally totters, even to complete dementia. The rectum and bladder act involuntarily. Bed sores form, and grave interference with the vital centres, or pulmonary affections soon put an end to a pitiful existence.

Whilst we have described three stages and sketched a clinical course, cases occur where the symptoms are chiefly ataxic, *plus* mental impairment; or mental, *plus* symptoms of disseminated sclerosis; but differential diagnosis or enumeration of the various types cannot be entered into here. Remember, however, that the optic discs *are not often affected*, and *wasting is rare*, except in the last stages, the patient often being fat, even to the end.

Treatment.—Put the patient under asylum treatment—*i.e.*, use common-sense principles, and by careful supervision, hygiene, and diet, try to prevent the disease progressing. *Rest, and firm, but gentle control*, do more good than drugs.

DISEASES OF PERIPHERAL NERVES. CRANIAL NERVES.

All have at least two origins—*viz.*, deep and superficial; all behind the 4th nerve may be said to have their deep origin in the 4th ventricle. The nerves of the special senses—*i.e.*, 1st (smell), 2nd (sight), 8th (hearing), have in addition a special connection with the so-called “centres,” situated in the cortex of the brain. We have left out the sense of “taste” here, because, although the glosso-pharyngeal carries the fibres for the posterior third of the tongue and the palate, there is no proof that it carries any taste fibres at its exit from the medulla, and there is increasing evidence in favour of the 5th being the nerve most implicated. Division of the 5th above the Gasserian ganglion results in paralysis of taste on the affected side, even when the glosso-pharyngeal remains intact (PURVES-

STEWART). For a special "sense" to be carried out properly, we can at once see that there must be a healthy continuity between *centre*, nerve *trunk*, and *special ending*; destruction of any of these will interfere with the carrying out of the function.

OLFACTORY OR FIRST PAIR.

Anatomy.—The true olfactory nerves issue from the nasal mucous membrane, and ascend through the cribriform plate of the ethmoid bone to the olfactory bulbs, which are in reality parts of the brain, although spoken of as the first cranial nerves.

Physiology.—The nerves of smell. Pungency should be carefully distinguished from the sense of smell, or the detection of odours; pungency is indeed due to painful stimulation of the 5th nerve in the *lower* part of the nose.

Clinical.—Highly nervous or insane people often complain of—

1. Hallucinations of Smell (*parosmia*).—They may complain of unpleasant odours, or that totally different odours are all alike—such as a rose and a herring having the same odour, which may be to them either pleasant or otherwise.

2. Increased Sensitiveness (*hyperosmia*).—Perfumes become intolerable, or patients may tell by odour alone the different members of their own family.

3. *Total or partial* loss of Smell (*anosmia* or *hyposmia*).—This may be due to—

- (1) Affections of the *terminations* of the nerve (most often associated with nasal catarrh or polypi).
- (2) Lesions of the nerve *trunk*.
- (3) Lesions of the *centre*, in the uncinate gyrus.

OPTIC NERVES.

Anatomy.—Commencing in the retina, the fibres pass backwards in the optic nerve through the choroid and sclerotic to

the optic foramen, and thence to the optic commissure, where the nerve terminates. Thus we get, on tracing backwards—

1. Optic Nerves.
2. Optic chiasma or commissure, where *partial* decussation takes place.
3. Optic tracts.
4. Ganglionic Centres. $\left\{ \begin{array}{l} \text{Optic thalamus.} \\ \text{External corpora geniculata.} \\ \text{Anterior corpora quadrigemina.} \end{array} \right.$
5. Cortical centres—*i.e.*, radiation in the occipital lobes.

The fibres from the inner side of each retina cross in the commissure to the optic tract of the opposite side; those from the outer part turn backwards into the tract of the same side (see diagram).

Physiology.—Nerves of sight.

Clinical—

1. Affections of the *terminations* in the retina include—
 - (a) Functional disturbances due to toxic effects, viz.—
 - (1) Uræmia.
 - (2) Jaundice.
 - (3) Drugs—quinine, santonine, etc.
 - (b) Hysterical amaurosis.
 - (c) Tobaccò amblyopia. The loss of sight is gradual, but, if the disorder persists, there may be *permanent* changes in the optic disc.
 - (d) Night blindness—(nyctalopia). Objects are clearly seen by day, but not in the dusk; or (hemeralopia), objects are *easily* seen in the shade, but, in the sunshine, with difficulty.
2. Lesions of the Nerve.
 - (a) Optic Neuritis.—The disc becomes blurred, swollen and red from congestion; there may be slight extravasations of blood; finally, there is an increase in the fibrous tissue, and atrophy of the nerve elements, causing often total blindness. It

is common in Bright's disease, tumours of the brain and cerebellum, and other intracranial affections.

The blindness is preceded by changes in three directions—

- (1) Diminished acuity of vision.
- (2) Alteration in the *field* of vision.
- (3) Altered perception of colour (Gowers).

There may be a considerable degree of optic neuritis before vision is materially interfered with.

- (b) *Primary Optic Atrophy* may occur in those who smoke too much, in tabes, sometimes in disseminated sclerosis and other nervous affections.

3. *Lesions of the Chiasma*.—If the lesion is confined to the central portion—*i.e.*, the decussation—the fibres passing to the *inner* or nasal portion of each retina will be involved, causing blindness of the *outer* half of each field—*temporal* hemianopia. If it is at the lateral part on one or other side it will cause a unilateral *nasal* hemianopia, and if there is such a lesion on both sides, a bilateral nasal hemianopia results. If the lesion is more extensive there may be total blindness of the eye on the same side, with temporal hemianopia of the opposite; or a more extensive lesion still may cause total blindness.

4. A lesion *behind the Chiasma*, whether in optic tracts, optic thalamus, corpora quadrigemina, or geniculate bodies, causes *homonymous hemianopia*, that is, blindness of the *temporal* half on the same side, and the *nasal* on the opposite side. A lesion in the occipital—

5. *Cortex* also causes homonymous hemianopia; but there is, in addition, word blindness, *i.e.*, the patient *can see* but fails to *read letters*. (See Aphasia.)

THIRD, FOURTH, AND SIXTH NERVES.

Anatomy.—The third nerve arises from the floor of aqueduct of Sylvius, passes through tegmentum of crus on the inner side, and forwards through the outer wall of the cavernous sinus to be distributed to—

1. The sphincter pupillæ.
2. Ciliary muscle.
3. All the muscles of the eyeball except superior oblique, and external rectus. *It also supplies the levator palpebræ superioris.*

Physiology.—The nerve concerned in accommodation of vision and certain movements of the eyeballs.

PARALYSIS.

1. Ptosis or drooping of upper lid.
2. Divergent squint, with inability to move the eye inwards, downwards, or upwards.
3. Loss of accommodation.
4. Paralysis of sphincter of iris = dilatation of pupil (*mydriasis*).
5. Double vision.

IRRITATION.

1. Convulsions or spasm of the muscles.
2. Convergent squint.
3. Contraction of pupil (*miosis*).
4. Great interference with field of vision.

Clinical.—Symptoms due to paralysis, or irritation of the third nerve, are common in many intracranial affections; for instance, the former condition is usually well marked in fractures of the base of the skull, the “*compression*” stage of meningitis, etc.; whilst *irritation* of the nerve is often brought about by tumours, the *first stage* of meningitis, reflexly by stimulation of the sensory fibres through intestinal irritation (worms, etc.).

It should be noted that only a *few* of the great number of fibres running in the third nerve may be affected, and the clinical phenomena will obviously vary with the number of fibres involved. We will consider the fibres to the iris first—

Paralysis of the Iris.—May be of three types—

1. Accommodative iridoplegia = failure to accommodate for near vision.
2. Reflex iridoplegia = failure of reaction to “light” stimulus, constituting the Argyll-Robertson pupil, a most important symptom in locomotor ataxia. In this case, the lesion probably

interrupts the fibres between the corpora quadrigemina and the centre for the sphincter iridis in the third nucleus.

3. Loss of skin reflex—failure of the pupil to dilate when the skin of the forehead is pinched.

Affections due to Alterations in the Muscular Apparatus of the Eye—

1. Nystagmus, or clonic convulsions of the muscles of both eyeballs. It usually results in rapid lateral movements, but the movement may be vertical. It ceases in sleep.

2. Strabismus or squinting.

Nystagmus may be due to—

1. Irritation of *fibres* in connection with the third nerve nuclei, as seen in—

- (1) Lesions of the restiform body in the medulla.
- (2) Lesions interrupting the afferent fibres administering to the functions of the cerebellum—*i.e.*, eighth nerve, which conveys impulses from the semicircular canals.

2. Tiredness or exhaustion of the ocular muscles after severe strain, common in miners after working with small Davy lamps; or after excessive glare, as seen in electric-light employees.

Fourth Nerve.—The nucleus of the fourth nerve is almost continuous with the lower end of that of the third. The nerve runs thence to the upper part of the roof of the fourth ventricle, where it decussates with the opposite fourth nerve. It then runs round the outer side of the crus to the base of the brain, and through the wall of the cavernous sinus to the orbit, ending in the superior oblique, which turns the eyeball downwards and outwards.

Sixth Nerve.—Arising from a nucleus in the floor of the fourth ventricle, it runs through the pons, and appears in the groove between the pons and medulla near the middle line. In the cavernous sinus it lies close to the internal carotid. It ends in the external rectus, which turns the eyeball outwards.

Fibres pass from its nucleus to the opposite third nerve and internal rectus.

Squinting or Strabismus.—In order to have perfect sight, there must be perfect harmony between all the ocular muscles; for instance, when we look at an object to the right, we turn *both* eyes in that direction, and thus we call into action the *external* rectus (sixth nerve) of the *right* eye, and the *internal* rectus (third nerve) of the *left* eye. In other words, on looking to the right side, we employ the services of two nerves, the third and sixth. Other movements will involve other combinations, such as the third and fourth nerves, third, fourth, and sixth, etc. Obviously, then, there must be a centre for the harmonious or co-ordinated actions of the third, fourth, and sixth nerves. This centre is placed by physiologists under the corpora quadrigemina, or in the “iter.” A failure of this harmonious action produces, on certain movements of the eyes, an alteration in vision termed *strabismus*, characterised by—

1. Diminished field of vision in certain directions.
2. Diplopia or double vision—*i.e.*, the appearance of a *true* and *false* image of the same object.
3. Erroneous projection—*i.e.*, errors in the judgment of the distance of objects (due to greater effort being required to focus for distant objects).
4. Secondary deviation—*i.e.*, if the affected eye be fixed on an object, it requires a greater effort than usual: but the muscle of the opposite eye, which acts in combination, will then be *unduly strained* and make a greater excursion than usual = secondary deviation.

RESULTS OF PARALYSIS OF SPECIAL OCULAR MUSCLES WHEN THE OTHERS ARE SOUND.

Paralysis of Rectus Superior: inability to raise eyeball properly above horizontal level; pupil may diverge somewhat downwards, and a little outwards (from action of the rectus inferior and the obliqui).

Paralysis of Rectus Inferior: inability to lower eyeball properly below horizontal level; pupil may diverge somewhat upwards, and a little outwards (from action of the rectus superior and the obliqui).

Paralysis of Rectus Externus (sixth nerve): inability to turn eyeball properly outwards; pupil diverges inwards (from action of rectus internus).

Paralysis of Rectus Internus: inability to turn eyeball properly inwards; pupil diverges outwards (from action of rectus externus).

Paralysis of Obliquus Superior (fourth nerve): but little alteration in movements of eyeball; slight deviation of cornea upwards and inwards, or simply upwards.

Paralysis of Obliquus Inferior: but little alteration in movements of the eyeball; slight deviation of the cornea downwards and inwards. (Paralysis of the sphincter of the iris, giving rise to a moderate dilatation of the pupil, and paralysis of the accommodation, often accompany this form of paralysis; this depends on the branch to the lenticular ganglion being given off from that branch of the third nerve which goes to the inferior oblique muscle. Occasionally, however, the lenticular branch arises from the sixth nerve.)—FINLAYSON'S *Manual*.

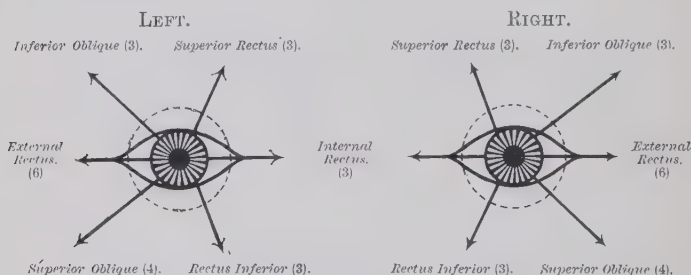


DIAGRAM to illustrate the directions towards which the *pupil* is moved by the separate action of the six muscles of the eyeball. The eyes are turned inwards and outwards by the external and internal recti; the internal rectus of one side is the yoke-fellow of the external rectus of the opposite side in these conjugate movements. The eyes are turned upwards by the superior rectus and the inferior oblique, downwards by the inferior rectus and superior oblique.

The eyes are represented facing the observer; if it be imagined that the eyes are looked at from the back, so that the diagram right-left becomes left-right, it will serve to illustrate the displacements in the field of vision caused by *paralysis* of individual muscles and nerves.—WALLER'S *Physiology*.

Strabismus is a most frequent cause of headache, etc., and probably many of the failures to treat headache successfully are due to the neglect of testing the visual apparatus, and thereby failing to rectify "eye-strain."

FIFTH CRANIAL NERVE.

Anatomy.—The fifth cranial nerve has a most extensive origin. The motor root arises from the floor of the fourth ventricle, and also from the outer wall of the aqueduct of Sylvius; the sensory arises from the Gasserian ganglion and ends in cells in the lower part of the pons and medulla. The nerve in front of the Gasserian ganglion divides into three sensory branches—

1. Ophthalmic.
2. Superior Maxillary.
3. Inferior Maxillary, with which the motor root becomes fused in the pterygo-maxillary region. The two first divisions, therefore, are entirely sensory; only the third is a mixed nerve.

By means of these branches, the fifth nerve has connections with four important ganglia—viz., the ophthalmic branch with the ophthalmic or lenticular ganglion, the superior maxillary with Meckel's ganglion, and the inferior maxillary with the otic and submaxillary or lingual ganglia.

The *ophthalmic branch* passes to the orbit through the sphenoidal fissure. It carries with it fibres from the sympathetic, which pass through the lenticular ganglion to innervate the dilator pupillæ. It is the sensory nerve of the skin of the upper eyelid and upper palpebral conjunctiva, forehead, front of the scalp to the vertex, mesial part of the skin of the nose; of the eyeball and lachrymal gland; and of the anterior and upper part of the nasal mucosa. The *superior maxillary branch* passes through the foramen rotundum to the infra-orbital canal, being connected in the spheno-maxillary fossa with Meckel's ganglion. From the ganglion the great superficial petrosal runs backwards to join the facial. The nerve supplies the skin of the cheek, upper lip, side of nose, lower eyelid and lower palpebral conjunctiva; the mucosa of the upper lip, upper cheek, upper jaw, hard and soft palate, uvula, tonsil, upper part of pharynx; the upper teeth, part of the nasal mucosa, and that of the middle ear. It contains some fibres of taste.

The *inferior maxillary branch* passes through the foramen ovale with the motor root. The motor and sensory parts become fused together, and the motor fibres of the trunk thus formed supply the temporal

muscle, the masseter, both pterygoids, the anterior belly of the digastric, the mylohyoid (muscles of mastication), and the tensor tympani. The sensory fibres supply the skin of the posterior part of the temple, part of the outer ear, the lower lip, lower cheek, chin, lower teeth and gums, tongue, floor of the mouth, and salivary glands. The lingual branch of the nerve is joined by the chorda tympani from the seventh. The auriculo-temporal is joined by a branch from the ninth (glosso-pharyngeal), which it sends to the parotid gland.

Taste Fibres.—The lingual branch of the inferior maxillary supplies the anterior two-thirds of the tongue. The taste fibres which it contains, however, pass from the lingual to the chorda tympani, which runs with the facial to the geniculate ganglion, and thence by the great superficial petrosal to Meckel's ganglion, where they enter the second division of the fifth. The glosso-pharyngeal supplies the posterior third of the tongue, but its taste fibres pass from it (through the petrous ganglion) by way of its tympanic branch (Jacobson's nerve) to the small superficial petrosal, and thence through the otic ganglion to the third division of the fifth. Section of the whole fifth nerve above the Gasserian ganglion destroys taste on the whole of the affected side.

Paralysis of the *whole* nerve causes—

1. Loss of sensation to parts supplied.
2. Paralysis and atrophy of all the muscles supplied, with R. D.

3. Diminished secretion of tears, absence of corneal reflex, and sometimes inflammatory changes in the cornea, due to—

(1) Anæsthesia (irritating particles are not felt, therefore not removed).

(2) Trophic changes?

4. Destruction of taste in affected side of tongue. The *fifth* is probably *the* nerve of taste, and the other nerves—viz., seventh and ninth—administer to taste *only inasmuch as they contain fibres from the fifth*.

5. Inability to detect pungent odours.

Trophic changes may be found in—

1. The cornea.
2. Hair roots (hair becoming grey).
3. Circumscribed areas of skin, affected with eruption of

herpes. The pain is often intense, and may persist for years after the herpes has disappeared.

Trophic changes are more often due to neuritic than to paralytic lesions.

Paralysis may be due to—

1. Disease of the pons.
2. Injury or disease at *base* of brain, medulla, etc.
3. Pressure from tumours, etc., on the branches as they pass through their foramina.
4. Peripheral neuritis.

A reference to the distribution of the nerve will give the symptoms attendant on paralysis of the whole or different branches.

Neuralgia may occur in all the branches of the fifth nerve (see *Neuralgia*).

Progressive unilateral atrophy of the face, a rare disease of childhood and youth, may be due to disease of the fifth, or to a sympathetic vasomotor affection.

SEVENTH NERVE.

The seventh or facial nerve arises from a nucleus in the lower part of the pons. It receives fibres from the nucleus of the third nerve, and emerges in the groove between the olive and restiform body. It enters the internal auditory meatus with the auditory nerve, passes through the Fallopian aqueduct, and leaves the skull by the stylo-mastoid foramen. At the geniculate ganglion, in the aqueduct, it is joined by the great and small superficial petrosal nerves. Within the aqueduct it gives off the chorda tympani (see *Fifth Nerve*). The nucleus is connected by motor fibres with the cortex in the fissure of Rolando. Briefly, its distribution is as follows :—

1. *Motor* to—

- (1) All the muscles of the face except the levator palpebræ.
- (2) Stylo-hyoid.

- (3) Posterior belly of digastric.
- (4) Stapedius.
- (5) Platysma myoides.

2. *Secretory* to—

- (1) Sub-maxillary gland.
- (2) Sub-lingual gland.
- (3) Glands of mouth and tongue.

3. *Sensory*.—In the Fallopian aqueduct it is associated with the chorda tympani, and a lesion there will consequently destroy the sense of taste in the anterior two-thirds of the tongue.

Paralysis Causes—

1. Motor changes—

- Face drawn to opposite side.
- Eye wide open, unwinking.
- Cheek puffs out with expiration.
- Food collects between cheek and teeth.
- Absence of wrinkles.

2. Sensory changes—

- Loss of taste in anterior two-thirds of the tongue, if the nerve is affected in the aqueduct.

Clinical.—Paralysis may be due to lesions affecting—

- 1. The cortical fibres—supra-nuclear paralysis.
- 2. The nucleus itself.
- 3. Nerve trunk in its tortuous course through the pons and bony canals, or after its exit from the stylo-mastoid foramen (Bell's paralysis).

The first (cortical) is associated with hemiplegia; and it should be at once noted that the paralysis will vary with the site of the lesion.

When it is a part of hemiplegia, the paralysis is on the *opposite side from the lesion*, that is, on the *same side* as the paralysed limbs; but the upper branches of the nerve escape, orbicularis palpebrarum is *not* involved, thus *movements of the eyelids are not affected—i.e., winking is possible.*

If the lesion be in the *lower* section of the pons, it will in-

volve the facial fibres *between* the nucleus and their emergence in the pons. Thus we get "*crossed paralysis*"—i.e., the face is paralysed on the *same* side as the lesion, the arms and legs (as before) on the opposite side.

Symptoms.—When the nerve is affected after it leaves the stylomastoid foramen (Bell's paralysis), the affected side is immobile, smooth, and expressionless. The eye cannot be shut, and usually waters freely. Smiling or attempted whistling, etc., brings out the contrasts markedly. Articulation of labial consonants is defective. The food collects between the cheek and teeth, because of the buccinator paralysis. The skin of the neck cannot be moved voluntarily (paralysis of the platysma).

If the affection is *within the aqueduct*, there are in addition loss of taste in the anterior two-thirds of the tongue (chorda tympani), and hypersensitiveness to sounds (*hyperacousis*) especially of low pitch, from paralysis of the stapedius.

If the nerve is affected between *the pons and the geniculate ganglion* there is no loss of taste, but deafness on the same side is almost always present from paralysis of the auditory nerve. If it is affected *in the pons*, there is no deafness or loss of taste, but there is usually paralysis of the sixth, as the facial fibres form a loop around its nucleus.

The electrical reactions are highly important. When there are *no polar changes, the case recovers rapidly*. If the reaction of degeneration be present, the prognosis is much more unfavourable. To sum up—

In the *nuclear or subnuclear* form of paralysis the whole of the one side of the face is involved, and typical "Bell's paralysis" results.

When of *cortical* origin it is—

1. Associated with hemiplegia.
2. *Upper part of face escapes.*
3. *Voluntary* movements are more affected than *emotional* movements.
4. The affected muscles seldom atrophy.

When in the lower portion of the *pons*, we get *crossed paralysis*, but of the nuclear type, as regards the muscles affected.

Causes.—

Cortical Form.—Those of hemiplegia.

Peripheral.—Exposure to draughts, rheumatic neuritis, disease of the middle ear, etc.

Nuclear.—Extension of chronic spinal diseases, etc.

* **Treatment.**—Remove if possible any pressure on the nerve by causing absorption of inflammatory products. Where the cause is syphilis, use specific remedies. Treat any ear disease, surgically if need be. In rheumatic cases, apply a blister over the point of exit of the nerve, and give diaphoretics and a smart purge. Maintain the tone of the paralysed muscles by a moderately strong continuous current applied for about a quarter of an hour several times daily, the positive pole being on the nape of the neck. The peripheral type is usually very amenable to treatment.

EIGHTH OR AUDITORY NERVE.

Anatomy.—It arises from two nuclei in the floor of the fourth ventricle. Some of its fibres are connected with the cerebellum, and also with the cortical centre situated in the superior temporo-sphenoidal lobe. The cochlear root is the true auditory nerve; the vestibular root the nerve of equilibration.

Physiology.—The nerve fulfils two functions—

Firstly, it is the nerve of hearing, or that by which sound undulations are conducted from the labyrinth to the cortex, and there analysed. The nucleus of the cochlear nerve is connected through the central auditory tract with the posterior quadrigeminal and internal geniculate bodies. Thence fibres pass to the cortical auditory centre.

Secondly, by means of its fibres in connection with the ampulla and semicircular canals it transmits impulses from the

oscillations of the endolymph in those regions, to the *cerebellum*, and thus assists the co-ordinating function in the maintenance of the equipoise of the body.

Paralysis causes total deafness, often attended by certain unpleasant subjective and objective symptoms.

Excitation of the auditory fibres causes unpleasant noises often accompanied by intense giddiness, nausea, and oscillations of the eyeballs.

It is highly important to remember the close sympathy and relation that exist between the optic, third, and auditory nerves; and again, between *these nerves and the cerebellum*.

Clinically we get—

1. Functional disturbances of the auditory nerve, including noises, buzzing, or “bell in the ear,” and these are often due to the accumulation of wax, altered tympanic pressure, etc.

2. Lesions affecting the cortical centre, causing word deafness—*i.e.*, spoken words are heard as noises, and not understood. (This condition may be associated with “word blindness,” which see.)

3. Lesions of the nerve at the base of the brain, causing permanent deafness. These are generally associated with lesions of the seventh nerve.

4. Disease of the labyrinth of the ear. The symptoms will depend on the extent of the lesion: there may be—

- (1) Hyper-sensitiveness.
- (2) Diminished hearing.
- (3) Total deafness.

Affections of the nerve cannot always be distinguished from those of the labyrinth, as both may cause “nerve-deafness.” This is tested by the absence of bone conduction of sound.

The subjective symptoms are typically brought out in—

5. *Ménière's Disease*.—An affection characterised by sudden and intense nausea, vomiting, vertigo, noises in the ear, and partial nerve-deafness of one ear. As we have already mentioned, nystagmus is frequently present. The attacks occur

paroxysmally. The symptoms are due to disturbance of intralabyrinthine pressure caused by

1. Hæmorrhage into labyrinth or semicircular canals.
2. Inflammation of labyrinth.
3. Pressure of basal tumours upon the nerve.

Treatment.—In any affection of this nerve first try to remove any discoverable cause. If associated with gout or syphilis treat these diseases primarily. Keep the external meatus clean, as deafness, giddiness, etc., are often caused by dried wax pressing on the drum. During an attack of Ménière's disease, the patient should be kept quiet in a darkened room; a brisk purge may be administered; and antipyrin, with an effervescing mixture containing caffeine, may be prescribed. Blisters over the mastoid process, repeated once a week, should be applied. Bromide of potassium in full doses is often useful. Salicylates may also be tried, or quinine, pushed to the stage of cinchonism. When the nerve is affected through disease of the bony canal, the treatment is highly unsatisfactory.

NINTH, TENTH, ELEVENTH, AND TWELFTH NERVES.

These nerves arise from nuclei in the lower part of the floor of the fourth ventricle, the twelfth near the middle line, the rest laterally. The eleventh is connected with the anterior cornu of the grey matter of the cord as low as the fifth cervical nerve. Its "bulbar part" really belongs to the vagus or tenth nerve, as it arises from a continuation of the same nucleus, whilst the "spinal part" (the true spinal accessory) arises from cells in the cervical cord. The ninth, tenth, and "bulbar part" of the eleventh (really part of the tenth) leave the side of the medulla behind the olivary body. The spinal accessory enters the skull by the foramen magnum. The twelfth leaves the front of the medulla in front of the olivary body. They all lie in the lower part of the posterior fossa of the skull. The twelfth enters the neck by passing through the anterior condyloid foramen, the rest through the jugular foramen. As they enter the neck they all lie between the internal carotid and the internal jugular vein, and are connected by communicating branches. The ninth and tenth are mixed nerves, the others motor.

The *ninth or glosso-pharyngeal* is a sensory nerve for the posterior third of the tongue, soft palate, fauces, and pharynx: motor for the middle constrictor of the pharynx and stylo-pharyngeus; inhibitory for movements of deglutition. The *tenth or vagus* supplies the pharynx, larynx, heart, lungs, œsophagus, stomach, and in part the intestines and spleen. It sends an auricular branch to the skin of the outer ear. Its lowest fibres of origin ("bulbar part" of the spinal accessory) are motor fibres for larynx and pharynx, and inhibitory fibres for the heart. Its pulmonary fibres are motor for the bronchial muscles, sensory for the respiratory passages. It is motor and sensory for the œsophagus, sensory for the stomach, partly motor for the stomach and intestines. The *eleventh or spinal accessory*, exclusive of its "bulbar part," supplies the sterno-mastoid and upper fibres of the trapezius. The *twelfth or hypoglossal* supplies the muscles of the tongue and depressors of the hyoid bone.

The *glosso-pharyngeal nerve* may suffer in disease at the base of the brain, thrombosis of the jugular vein, tumours and injuries external to the skull. Isolated paralysis of this nerve has not been observed in man. If it is affected above the petrous ganglion, anæsthesia of the pharynx and back of the tongue, and dysphagia, are observed; if at or below the ganglion, also loss of taste in the posterior third of the tongue.

The Vagus Nerve may be implicated in diseases of the cranial bones, the meninges, and in vertebral aneurism. In the neck injuries, tumours, or aneurisms; in the thorax mediastinal tumours, or aneurisms, may compress the nerve. Its recurrent laryngeal branch may be compressed on the right side by pleuritic apical adhesions, on the left by aortic aneurisms. The nerve may be paralysed in alcoholic paralysis, poisoning by lead or arsenic, after diphtheria or influenza, in diseases of the medulla and disseminated sclerosis.

Symptoms vary with the seat of the lesion. Intracranial lesions may affect all the roots, or only the upper or lower. The hypoglossal is often also affected. If the whole nerve is implicated, there is unilateral paralysis of the soft palate, fauces, and larynx, with laryngeal anæsthesia. If both nerves are implicated the paralysis is bilateral and there are in addition tachycardia and cardiac irregularity; slow, irregular respiration; gastric pain and vomiting, and sometimes loss of the sensations

of hunger and thirst. For details of the laryngeal paralysis see p. 298.

The *spinal accessory nerve* is affected by diseases at the base of the brain, it may suffer in disseminated sclerosis, and in the neck it may be pressed upon by tumours or abscesses. It is sometimes attacked by "rheumatic" neuritis. Weakness and wasting of the sterno-mastoid and upper part of trapezius, with R. D., are the result. The chin is rotated towards the opposite shoulder, and the shoulder on the affected side can be only imperfectly raised.

The *hypoglossal* suffers more frequently within than outside of the cranium. Weakness and wasting of one half of the tongue, with R. D., are the chief symptoms. The mucous membrane of the tongue is wrinkled, and thrown into longitudinal folds. In facial hemiatrophy (see Fifth Nerve) one half of the tongue may atrophy, but there is no R. D.; in hemiplegia there is lingual paralysis, but neither atrophy nor R. D.

SPINAL NERVES.

The Phrenic Nerve, arising chiefly from the fourth cervical root, with fibres from the third and fifth, supplies the diaphragm, and sends fibres to the pleura, pericardium, inferior cava, and right auricle. It may be affected in fracture of the cervical vertebræ, spinal hæmorrhage, and tumours of the cord; in the neck by wounds, tumours or aneurism; in the thorax by tumours, aneurism, or pleural affections. It may be involved in peripheral neuritis.

Symptoms.—When both nerves are affected there is bilateral paralysis of the diaphragm. The epigastrium sinks in during inspiration, and the movements of the upper part of the thorax are excessive. Dyspnœa occurs on the slightest exertion. Respiratory diseases are rendered more grave by the existence of this condition.

Unilateral paralysis can only be recognised by careful comparison of the movements of the two sides.

Prognosis is good in rheumatic and post-diphtheritic paralysis, bad in central lesions.

Treatment.—In paralysis due to central lesions, treat the lesion ; in peripheral paralysis stimulate the nerve by electricity, placing one pole deeply to the outer side of the clavicular head of the sterno-mastoid, the other over the epigastrium.

Paralysis of the other spinal nerves need not be referred to in detail. A knowledge of their anatomical distribution and function should enable us to reason what to expect.

NEURITIS.

Inflammation of a nerve or of its fibrous sheath.

Ætiology.—Sometimes of obscure origin ; sometimes due to exposure to cold, traumatism (as when an intoxicated man “overlies” his brachial nerve), spreading of contiguous inflammation, or pressure by tumours. Predisposing causes are gout, syphilis, *diabetes mellitus*, and also poisoning by lead or arsenic.

Morbid Anatomy.—In simple neuritis the sheath of the nerve and the interstitial substance are more affected than the fibres, which suffer only secondarily. The ordinary inflammatory changes are present.

Symptoms.—Vary with the special nerve affected. There may be disturbed sensibility, pain in the peripheral distribution of the nerve, or tenderness in its course. Where a motor nerve is involved, twitchings of muscles and rapid atrophy follow, along with paralysis of function. If trophic fibres are involved there may be “glossy skin,” œdema of the extremities, herpes, etc.

Treatment.—Treat any specific cause by appropriate remedies ; locally leeches, fomentations, or flying blisters. If there be paralysis, galvanism or massage.

MULTIPLE NEURITIS.

Inflammation affecting simultaneously or in rapid succession nerves in different parts of the body.

Ætiology.—Due principally to the action of poisons circulating in the blood on the terminations of peripheral nerves. These poisons are of various classes :—

1. Extrinsic : alcohol, arsenic, bisulphide of carbon, lead.
2. Microbic : as in the specific fevers, septic states, beri-beri, malaria, influenza, etc.
3. Due to metabolic changes ; as in diabetes, gout, syphilis, etc.

General malnutrition is responsible for cachectic and senile cases.

Morbid Anatomy.—The changes are most extensive at the peripheral terminations of nerves, and are usually symmetrical. They are both inflammatory and degenerative, but often only visible under the microscope. The degenerative changes affect the nerve fibres, in which the axis cylinders are atrophied and the medullary sheath broken up. The inflammatory changes are seen in the interstitial tissue, and are much less marked.

Alcoholic paralysis is one of the best examples of multiple neuritis, and will therefore be described as the typical form.

Ætiology.—It usually occurs amongst *secret* drinkers, and for that reason women are more often attacked. Most cases occur after thirty-five years of age. Club-men and brewers furnish a number of cases.

Symptoms.—We must remember that the manifestations of chronic alcoholism differ much in individuals ; there are at least four well-marked types.

1. That in which tremor predominates.
2. The anæsthetic type.
3. The paralytic type.
4. The ataxic form.

First there is tingling like pins and needles in the muscles generally, then numbness of the fingers and toes; vasomotor spasm causing dead fingers, and muscular cramps. Progressive paralysis compels the patient to take to his bed; the extensors are more implicated than the flexors, and consequently the patient lies with dropped feet and dropped wrists (the feet generally first). Other muscles are gradually involved, and phonation may be lost or much weakened. Atrophy of the affected muscles rapidly supervenes, being especially well marked in the extensors below the knee and elbow. Partial or complete reaction of degeneration is present. There is loss or diminution of the tendon reflexes. The atrophied muscles and the nerve trunks are often exquisitely tender. There are therefore anæsthesia to touch and hyperæsthesia to pain.

The mental state often suffers: the memory is almost lost, and often delusions are marked features. The ataxic form resembles tabetic ataxy in the numbness of the feet, absence of knee-jerk, locomotion, and non-wasting of the anterior tibial muscles; but, in such cases the gait is different from the true ataxic walk, as the patient not only throws out *his feet brusquely*, but *steps high in order to clear his toes of the ground*. (Dr. FERRIER.) “Alcoholic pseudo-tabes” is, however, rare.

Treatment.—Cut off the supply of alcohol entirely. Perfect rest in bed. To ease the pain—iodide of sodium, antipyrin, or caffeine may be given, and hot applications locally. The paralysis should be treated by strychnine hypodermically, and a mixture of strychnine and capsicum may help to relieve the alcoholic craving. Insomnia may be treated by bromides or trional. The gastric symptoms of alcoholics require careful dieting. General tonic treatment is also necessary. Afterwards—good feeding, massage, and electrical stimulation as a rule will, if persevered in, cure.

In all cases of multiple neuritis, the special cause must be carefully sought out, and eliminated by appropriate treatment as speedily as possible.

NEURALGIA.

This term means paroxysmal pain in the area of distribution of a sensory nerve, usually without discoverable structural change, although in other cases such changes have been noted. It is commonest in adult life, and affects males more than females except during pregnancy and at the climacteric. It follows upon debilitating diseases, exposure to cold and damp, toxic influences, peripheral irritation (carious teeth, errors of refraction, etc.). The chief symptom is paroxysmal pain, slight or intense, arising spontaneously or from local stimuli, lasting a few seconds or several minutes, and sometimes recurring many times a day. Reflex motor spasms are frequent. Secretory disturbances (lacrymation, etc.) and vasomotor or trophic changes may occur. There are usually "tender points" over the points of exit of nerves through bony foramina, the points where they cross bones or fasciæ, and the corresponding vertebral spines.

Trigeminal Neuralgia or "*tic douloureux*" is very common and very severe. It may affect all the divisions of the fifth nerve, but much more usually one or two, most frequently the ophthalmic. It is almost never bilateral. Exciting causes may be found in the mouth, nose, or eye. The symptoms are sudden onset of almost unbearable pain in the area of the affected branch, from which it may spread to other branches, involuntary spasm of the facial muscles, lacrymation, excessive secretion of nasal mucus, or salivation. During the attack the hand is pressed upon the affected part; in the intervals even a touch may set up the pain, or it may follow movement of the facial muscles, or washing the face. In long-standing cases the hair may fall out or become grey. "Tender points" are to be found at the exit of the branches from their bony foramina.

Other forms of neuralgia—cervico-occipital, intercostal, mammary, sciatic, etc.—require no special description here.

Treatment.—Remove any causes of peripheral irritation. Improve the general health by tonics and massage. In recent cases diaphoretic treatment—hot air, hot baths, etc.—is often useful. Quinine or gelsemium may be pushed till toxic symptoms appear. Relieve pain by one or other of the anti-pyrin group, salicylates, or iodides. If necessary give morphia hypodermically, but do not allow the patient to treat himself thereby. Galvanism, the positive pole being over the tender point, is sometimes of use. The most successful drug in trigeminal neuralgia is gelsemium, but it must be pushed. Surgical treatment, in the last resort, comprises nerve stretching and nerve excision, from which relief is usually temporary, and excision of the corresponding ganglion—in the case of the fifth nerve the Gasserian ganglion.

FUNCTIONAL DISORDERS OF THE NERVOUS SYSTEM.

MIGRAINE.

(HEMICRANIA. SICK HEADACHE.)

AN affection characterised by paroxysmal headache, nausea, and vomiting, and sometimes preceded by disorders of vision. It usually begins about puberty, rarely after thirty, and is somewhat more common in women. Hereditary tendency is very marked, and allied neuroses are often seen in other members of the family. An exciting cause for the first attack is often undiscoverable, but worry, overwork, disturbances of the general health, and especially eye strain, predispose to repetitions. The condition is probably to be accounted for by derangement of the cells of the cerebral cortex.

Symptoms.—An attack usually begins with headache, but may be preceded by depression or visual disturbance. The pain, which begins over the forehead, very often in the early morning, is at first slight. It soon becomes severe and boring, is localised just above one eyebrow, and spreads thence till it involves the whole side of the head, and sometimes the neck and arm. The nerves of the scalp are tender on pressure. The attack lasts twelve to twenty-four hours or more; it is accompanied by increasing nausea, and ends in vomiting, which brings relief. In about half the cases there are visual disturbances either before or during the attack. Sometimes

merely flashes of light may be noted, in other cases there may be partial or complete homonymous hemianopia, with "spectral" bright spots appearing in the blind side of the field of vision. The disposition to migraine tends to pass off about the end of the first half of life.

Treatment.—In the attack, give a smart saline aperient at once. Absolute rest and quiet in a darkened room, warmth to the feet, evaporating lotions to the head, antipyrin or phenacetin, are the other elements of treatment. In the intervals, improve the general health by attention to stomach and bowels, correct eye strain, remove adenoids, etc. Tonics such as iron or strychnine may be given. A prolonged course of bromides with tincture of gelsemium often does much good.

EPILEPSY.

A chronic paroxysmal affection characterised by sudden attacks of unconsciousness, with or without convulsions, and often by a tendency to mental deterioration. Attacks without convulsions are known as minor fits (*petit mal*), those with convulsions as major fits (*grand mal*).

Jacksonian epilepsy differs so much in ætiology, treatment, and pathology, that it will be dealt with by itself.

Ætiology.—The disease frequently begins in early childhood; probably one-fourth of the cases begins before the child is ten years of age, and *three-fourths* between ten and twenty years of age. *When it begins in adults* look for a *local* cause; such cases may be of a *Jacksonian or hysteroid type*, or due to alcoholism or chronic nephritis.

Heredity is a most important factor, in so far as children of families in which there is a history of insanity, hysteria, alcoholism, neuralgic affections, etc., are prone to this affection.

Exciting Causes.—Those usually assigned by relatives probably merely determine an attack, but do not cause the disease. Frights and falls or injury to the head are often mentioned.

Reflex irritations as from a tight prepuce, masturbation, worms, dentition, are also blamed.

Given a person *born with a marked and distinctly neurotic temperament*, it is evident that any undue cause of nervous irritation, ranging from a tight foreskin to absolute debauchery, must be a potent factor in maintaining the nervous instability which predisposes to epilepsy.

Pathology.—The structural changes which have been found are purely secondary. Reasoning from what we know through direct experiments on the cortex, the great symptoms of epilepsy—*i.e.*, loss of consciousness, convulsions, and coma—leave us in no doubt that the higher centres of the cortex are affected. Should an epileptic subsequently become hemiplegic from a lesion in the internal capsule, the convulsions do not affect the paralysed limbs, showing that they originate in the cortex, impulses from which are now cut off. Convulsions may be produced by excitation of any part of the cortex, and the nature of the aura may help to localise the seat; visual aura probably = occipital lobe, auditory aura = temporo-sphenoidal lobe, motor aura = Rolandic area, etc. The actual muscular spasm is due to the sudden violent action or “discharge” of the cortical grey matter.

Symptoms.—

Grand mal—divided into three stages.—

1. *Aura* or warning. Vertigo is a very common form. The aura may be referred to the stomach (sinking or nausea), or to the heart (palpitation); it may be visual or auditory; it may be sensory—numbness, “pins and needles,” etc., beginning distally and mounting up the limb—or motor—twitchings or contraction. The aura of idiopathic epilepsy, in contrast to that of the Jacksonian form, is usually very brief.

2. *Fit*.—

(a) The patient drops down suddenly after uttering a piercing shriek (epileptic cry). The cry is sometimes absent.

(b) *Tonic Stage*.—The head and eyes are *turned to the side* on which the convulsions are stronger, the hands clenched, the elbows flexed, the legs rigid and extended, and the muscles of the chest by their contraction interfere with the respirations, so that the preliminary pallor is succeeded by lividity. The pupils are dilated, the conjunctival reflex lost, eyes fixed, and sensibility is abolished. This stage lasts from thirty to forty seconds, and then passes into the—

(c) *Clonic Stage*.—Convulsions of the face quickly extend to all the muscles of the body; the tongue may be pushed between the teeth and bitten severely; and the foam at the mouth is then coloured with blood. The face assumes a purplish hue, and the eyes seem to protrude from their sockets. Urine, semen, or fæces may escape involuntarily, and the pulse is often much embarrassed by the muscular contractions. This stage lasts about four minutes or more, and then the convulsions cease. The breathing gradually becomes easier, the features regain their natural colour, and consciousness may be quickly recovered; or, what is more common, the patient may pass into the third stage, namely,—

3. *Post-epileptic Condition*.—The patient falls into a comatose state, succeeded by a natural sleep. Rarely in this form are there post-epileptic maniacal manifestations.

During the attack the superficial and deep reflexes are abolished, while for a short time after it they are exaggerated. There is commonly a large excretion of urine after the fit. In the attack the temperature is slightly raised. One attack may be rapidly followed by another, with no recovery of consciousness in the interval, and this condition may persist for some hours (“status epilepticus”). In such cases the temperature rises even to hyperpyrexia, and death may result from exhaustion.

Petit mal, or minor epilepsy.—

In this form the patient is suddenly seized with unconsciousness, the eyes become fixed, speech momentarily incoherent, but there are *no convulsions*. The attacks vary much in

severity. Sometimes they are so slight as not to be noticed by others than the patient. This form of epilepsy must be remembered, for though the fits are often trifling, yet, the "post-epileptic" condition is sometimes of a very serious nature: two such forms are described—

Post-epileptic conditions—

1. A condition in which the patient performs automatic actions, of which he is then and *afterwards entirely unconscious*. A father may kill his child; well-to-do patients may steal; etc.

During this state the memory of *past* events remains, and thus people whom the sufferer dislikes may be attacked or killed, apparently deliberately.

2. A condition characterised by maniacal manifestations, intense passion, etc.

Prognosis.—Frequency of epileptic attacks varies considerably. Females are more frequently attacked during a menstrual period. Epileptics may be fairly strong between the attacks, and finally recover completely; but too often some mental weakness shows itself in a permanent form. Imbecility has developed after a few fits in children. The dangers during the fit must not be forgotten—fatal falls, burns, or other injuries are common.

Diagnosis.—Must be distinguished from hysteroid convulsions, uræmia, malingering, etc.

	EPILEPSY.	HYSTERIA.	MALINGERERS.	URÆMIC CONVULSIONS.
<i>Consciousness.</i>	Lost.	May be partially lost.	Normal.	Lost in later stages; coma is prolonged and deep.
<i>Pupils.</i>	Dilated during fit.	Normal.	Normal.	First contraction, followed by dilatation.
<i>Tongue.</i>	Often bitten.	Normal.	May be bitten to simulate real fit.	Normal.
<i>Restraint.</i>	Necessary to prevent accident.	Necessary to control violence.	Not necessary.	Not necessary.
<i>Onset.</i>	Rapid and sudden, patient falls unconscious.	Usually after some mental excitement, patient may fall into a "dazed" condition.	Always under conditions where the fraud may hope to gain sympathy. He falls in a business-like manner, taking care not to sustain painful injuries.	Preceded by alterations in health, urine, etc. Patient is usually in bed before convulsions come on.
<i>Duration.</i>	A few minutes.	Much longer.	Variable.	Prolonged.
<i>Recovery.</i>	Moderately rapid.	Very variable.	Very rapid after object has been gained.	Slow, if not fatal.

Treatment.—Try to discover any cause of reflex irritation and remove it if possible. Try to keep the general health as perfect as possible. Dangerous occupations must be avoided, and bathing forbidden. Stimulants are not allowed. Of drugs, bromides stand first; no permanent ill effects have been definitely traced to a very prolonged use of these drugs. They may be combined with arsenic to prevent the bromide rash. Careful attention must be paid to the state of the bowels, menses, etc.

During the Fit.—Loosen the collar, corset, etc., and put something between the teeth to prevent the tongue being bitten; inhalation of nitrite of amyl; dash cold water on face, etc.; and prevent patient from injury through his convulsive movements. If there is a well-defined aura, as numbness or twitching in one hand or foot, the attack can often be prevented by prompt ligature of the limb above. If

the attacks are nocturnal, the patient should sleep on a hard bed, and use a hard pillow. During the attack he tends to turn on his face, and may be asphyxiated in the post-epileptic coma. Gowers states that when a person is found dead in bed, and lying on his face, there is very strong presumption of death after convulsion.

In the status epilepticus a full dose of bromide (gr. 40-60) or chloral (gr. 40) should be given by the rectum, or hyoscyne hypodermically. Chloroform is useful to check the convulsions until other drugs have time to act.

JACKSONIAN EPILEPSY.

Jacksonian epilepsy is an affection characterised by epileptiform convulsions, often unattended with loss of consciousness, and dependent on a coarse lesion of the cerebral cortex.

Ætiology.—It is usually associated with syphilis, and therefore most frequently seen amongst adult men; but cortical fits may be due to other tumours, to abscesses, meningitis (especially pachymeningitis hæmorrhagica), and injuries.

Symptoms.—First twitchings of a muscle or group of muscles in the arm, leg, or face. The spasms may be distinctly localised, and the patient, being conscious, often watches the progress of the march, and thus affords most important information, which often enables the physician to localise the lesion. The convulsions very rarely become general, but often implicate the whole of one side. They are seldom painful. Consciousness may be lost late in the fit, which is usually followed by a transitory paralysis of the affected muscles or side.

Diagnosis.—The slow onset, absence or late onset of unconsciousness, and the localised type of the convulsions sufficiently distinguish them from ordinary epilepsy. *The part which first shows rigidity during the convulsion points toward the motor centre for that part as the seat of the greatest irritation.*

Sensory disturbances precisely analogous to the motor fits may also occur, or may precede them as an aura. They are of equal localising value.

Treatment.—

1. If syphilis be the cause, give large doses of iodide of potassium and mercury.

2. If not syphilitic, then trephine, explore, and remove if possible any discoverable source of irritation. Bromides may be used as in epilepsy.

INFANTILE CONVULSIONS.

Causes.—The instability, and especially the reflex excitability, of the incompletely developed nervous system of the child, aided by such predisposing causes as—

1. Rickets. The chief exciting cause in this condition is reflex irritation, especially from gastro-intestinal irritation, and teething; round-worms, otitis media, and phimosis are also occasional reflex causes.

2. Infectious fevers. Convulsions take the place of the rigors of the adult.

3. Cerebral congestion or asphyxia, well seen in severe cases of whooping-cough, and after exposure to the sun in a perambulator, etc.

4. Less often cerebral diseases, as at the onset of infantile hemiplegia or diplegia, and meningitis.

5. Congenital syphilis.

6. Hereditary predisposition.

Infantile convulsions are most common before the eighteenth month. They may recur frequently and end in the establishment of epilepsy. The symptoms are less severe than those of true epilepsy, though the fit may last longer. The convulsions are bilateral, but often not universal.

Treatment.—During the fit:—A few whiffs of chloroform, warm bath, ice to head, small doses of chloral by the rectum,

etc. After the fit:—A dose of grey powder or calomel, saline laxatives, and bromide in appropriate doses for a week or more. Remove any source of peripheral irritation ; attend to bowels, diet, fresh air, and treat the general condition.

TETANY.

A condition characterised by tonic muscular spasm chiefly in the distal parts of the limbs, with increased excitability of muscles and nerves.

Ætiology.—Probably due to a toxin or toxins acting on the peripheral motor neurons. In childhood, males, in adult life, females, are more often affected. In children, rickets is almost always present ; in adults, the disease occurs in connection with pregnancy and lactation, gastric dilatation, and gastro-enteritis, extirpation of the thyroid, and after specific fevers.

Symptoms.—Spasm commences in the hands, then attacks the feet, and may extend to the arms and legs, or even the trunk and face. The thumb is flexed and drawn into the palm, the fingers are flexed at the metacarpo-phalangeal joint, extended at the others, and adducted, so that the hand becomes cone-shaped (“accoucheur’s hand”). The wrist and elbow are flexed, and the shoulder adducted. The wrist is drawn to the ulnar side, and the forearm is carried across the chest. In the feet the toes are flexed, especially the great toe ; the foot is inverted and the ankle extended. If the trunk be affected there is usually emprosthotonos, less often opisthotonos. The spasm may last from a few minutes to a few days. It passes off slowly, and recurs after irregular but brief intervals. It is usually painful, and passive movement increases the pain. Between the spasms tapping on a muscle or its motor nerve sets up a brisk contraction (*Chvostek’s sign*). Pressure on a nerve trunk, or compression of the limb impeding the circulation, will set up a spasm (*Trousseau’s sign*). The electrical reactions, galvanic and faradic, are increased in intensity.

In *tetanus* the spasm is more continuous, the muscles of

the jaw are early implicated (trismus), and the fingers and hands escape.

Treatment.—Remove if possible any evident cause. Improve the general health, and keep the patient at rest. For drugs, bromides and chloral.

HYSTERIA.

Hysteria is a form of functional affection of the highest nervous centres, leading to emotional disturbances without failure of intellect, and to motor and sensory changes without organic disease of the brain or cord.

Ætiology.—It is seen principally, but not exclusively, amongst women, and has long been considered symptomatic of disorders of the ovaries or uterine functions. This view is still much too prevalent, especially among the laity, but there is no doubt that such conditions may lead indirectly to hysteria by the production of a depressed mental state. It may be the result of nerve exhaustion from any cause. Hereditary instability of the neurons is one of the most important factors. Injudicious treatment by relatives is also important. The patients are chiefly young adults. The exciting causes, in those predisposed to the affection, are chiefly psychical,—fright, shock, grief, etc. It must not be forgotten that hysteria and organic disease of the nervous system may co-exist, and that the symptoms of the former may for a time mask those of the graver affection.

Symptoms.—They are too varied to tabulate completely. Indeed, it is difficult to say what symptoms may not be hysterical in a given case. The more common phenomena are—

1. Disturbances of the sensory apparatus.—

- (1) Anæsthesia of localised areas, or complete hemianæsthesia and hemianalgesia. The distribution never corresponds accurately to that of spinal segments, nerve roots, or nerve trunks. Complete

hemianalgesia is excessively rare in *organic* hemianæsthesia. There may be unilateral loss of one or other special sense, *on the same side*. Anæsthesia may disappear during sleep.

- (2) Hyperæsthesia of skin or special senses.
- (3) Loss of muscular sense.
- (4) *Globus hystericus*, a sensation as of a "ball of wind" rising from the epigastrium to the throat.
- (5) Neuralgias. "Tender points" over vertebral spines, ovarian region, etc. Pressure on these may excite a convulsion ("hysterogenetic points").

2. Disturbances of the motor apparatus.—

- (1) *Paralytic*. Usually paraplegia of rapid onset, without wasting or electrical change. It may be partial or complete. Knee-jerks exaggerated, *ankle-clonus absent*. Sphincters unaffected. Hemiplegia or monoplegia may occur, also aphonia.
- (2) *Spastic*. Contractures of a limb or segment of a limb, entirely flexor in the arms; in legs, toes flexed, foot dropped and inverted, knees and hips extended. Contracture of abdominal muscles may cause "phantom tumour."
- (3) *Convulsive* (hystero-epilepsy). After an aura, tonic stage (opisthotonos), followed by leaping or struggling with purposive movements. Screaming, talking, or singing. Consciousness impaired, not lost. Tongue never bitten.
- (4) Tremors or inco-ordinate movements.

3. Visceral Disturbances.—

- (1) Palpitation.
- (2) Vicarious menstruation.
- (3) Vomiting, belching, etc.

4. Psychical disturbances.

Morbid desire for sympathy, leading to wilful additions to the real symptoms,—heating the thermometer, factitious skin eruptions, etc.

Apathy and obstinacy.

Sudden transition from the joyous to the sad.

Somnambulism.

Nymphomania.

Catalepsy—may simulate death.

Melancholia, mania, etc., may occur as associated psychoses, but are not characteristic of hysteria.

5. Vasomotor disturbances.

It will be seen from the above table that it would not be easy to mention what symptoms may not crop up in various forms of hysteria. So closely does hysteria simulate real disease that the best diagnosticians have been deceived. When grave symptoms appear, even after a careful consideration of the history, etc., of the patient, a *provisional diagnosis* should be made until the patient has been for some time under treatment (Dr. Ranny's *Lectures*). It is nevertheless possible to make the diagnosis in the majority of instances. Points in favour of hysteria are (1) the abrupt appearance and disappearance of symptoms after trifling causes; (2) a collocation of symptoms not explicable by an organic cause; (3) absence of symptoms to be expected in organic disease (optic neuritis, incontinence of urine, ankle-clonus, etc.).

Treatment of hysteria depends on the type. All forms, however, require *firm*, kind, and judicious treatment. The physician must first gain the patient's full confidence, and having done this he may use bromides, tonics, massage, and "Weir-Mitchell" treatment. The various special symptoms must be treated on general principles. It is doubtful if such drastic measures as excision of the clitoris, removal of the ovaries, or applications of the actual cautery are admissible, though they have been practised by eminent authorities.

NEURASTHENIA.

By neurasthenia are meant the manifestations of nerve exhaustion, leading to altered bodily nutrition. It is often

difficult to distinguish between hysteria and neurasthenia, but there are some important differences.

NEURASTHENIA.

1. Occurs most often in men.
2. Usually directly attributable to overwork.
3. Little desire for sympathy.
4. Usually wasting is present.
5. Very amenable to proper treatment.

HYSTERIA.

1. Women most frequently.
2. Often seen amongst the indolent and the rich.
3. Great desire for sympathy.
4. Often plump or fat.
5. Anything but amenable to treatment.

Prolonged overwork, especially if associated with worry, or lasting grief, is the chief cause, but sudden shock may give rise to the disease, which is very frequent after railway accidents ("railway spine"). Drug habits, especially the morphine habit, may produce the symptoms, or aggravate them if present. They may also follow *influenza* or enteric fever. Alcoholism is an indirect predisposing cause.

Symptoms.—The patient is of spare build, or distinctly wasted. His appetite is variable, his bowels irregularly costive or loose, and he suffers from insomnia or broken sleep. He is much depressed and broods continuously on his symptoms, which afford him a melancholy joy. He lacks concentration, and is unfit for mental work. He is restless, and unduly irritated by loud sounds or bright lights. He dwells upon his visceral sensations. Headache, tinnitus aurium, dimness of vision, and spinal irritation are frequent symptoms. Palpitation, præcordial pain, tachycardia, and vasomotor disturbances are common. The muscles are flabby, and may be wasted. Fine tremors in the hands are often present. The knee-jerks may be exaggerated. Dyspepsia is frequent, and flatulence very marked.

Treatment.—The causes and definition of the disorder make it sufficiently obvious what line of treatment should be employed; indeed, it may be summed up in four words—complete change, rest, tonics. The principles of treatment are the same as those for hysteria.

CHOREA OR ST. VITUS'S DANCE.

A disease most common in childhood, characterised by spontaneous irregular movements of the limbs or the whole body, with a tendency to endocarditis and other rheumatic phenomena.

Ætiology.—It is most frequently seen amongst female children between five and ten years of age, and also during the adolescent period. Girls are attacked three times more frequently than boys. It hardly ever occurs before the age of three. Emotional or excitable children are prone to this affection, and a neuropathic tendency is often traceable in the family history. Clinical records show a more or less constant relationship between chorea, endocarditis, and rheumatism. Although there are cases in which neither a history of rheumatism nor endocarditis can be traced, the *family* history very constantly reveals evidence of rheumatism. In the adult, chorea may occur during pregnancy, but chiefly in the first pregnancies of unmarried girls, where the influence of emotion counts for much.

The **Exciting Causes** are mainly emotional—

1. Disturbed mental conditions, brought about by hereditary weakness.

2. Fright or shock.

3. Excessive study or overstrain at school. This is more apt to aggravate an existing chorea than to cause one. Eye strain is of only secondary importance.

Imitation of choreic movements in another, formerly considered important, is of little or no significance. "No cases have arisen in this way at the Great Ormond Street Hospital during thirty years" (Colman and Collier).

Pathology.—No constant lesion is found in chorea. Many observers have noticed punctiform hæmorrhages in the cortex, and also embolic plugging of the small capillaries of the basal ganglia, with small foci of softening; but such conditions are

infrequently found. They led to the theory that chorea is caused by detached bits of endocardial vegetations which become arrested in the cortical vessels, and there set up irritative changes. Endocarditis, however, is by no means invariably present, and this view is now generally abandoned. Various micro-organisms, chiefly streptococci and staphylococci, have been found in the cerebral cortex and cerebrospinal fluid, and Poynton and Payne have isolated a diplococcus which produces chorea in animals, and can be recovered from their cortical arterioles. The modern view is therefore that chorea is of microbic origin, and is to be attributed essentially to "inherent instability in the sensorimotor sphere, together with a toxæmia or a shock sufficient to disarrange the customary association- or contact-areas in the cortex, basal ganglia, and cord." Most probably both the endocarditis and the chorea are the result of the circulation in the blood of some unknown toxin.

Symptoms.—

1. Are irregular and purposeless convulsive movements of hands, facial muscles, etc., rendered worse on attempting to execute voluntary acts, or fine movements—such as picking up pins, conveying a cup to the lips, writing, or touching a particular point, etc. Dr. Sturges sums up the symptoms as "an extremely exaggerated fidgetiness." The movements cease as a rule during sleep, but may be so violent as to prevent it. The condition may affect one side only (hemichorea).

2. Combined with the irregular movements there is inco-ordination, manifested in the movements of writing, in unsteady gait, and in irregular movements of respiration, the diaphragm and intercostal muscles failing to act together. Muscular weakness is a later symptom as a rule.

3. Characteristic attitude when in bed—child lies extended in bed, twisted to one side (pleurosthotonos), and then changes suddenly to an exactly opposite condition.

4. Irregular and rapid action of the heart, sometimes amounting to "delirium cordis."

5. Soft systolic murmurs at the apex of the heart, due to endocarditis, or hæmic murmurs at the base.

6. Altered mental conditions are sometimes most marked; the patient laughing or crying in turns on being spoken to. Increased irritability is common.

An ordinary case lasts from one to three months. Recovery is always gradual. Relapses are common.

Treatment.—

1. *Rest in bed* is the first and essential point of treatment.

2. Arsenic in increasing doses, cod-liver oil, abundant food, and careful attention to the bowels. Antipyrin or phenacetin in increasing doses is also useful. Salicylates are of value only in rheumatic complications.

As soon as convalescence seems established, allow the patient plenty of fresh air and gymnastic exercises.

SPASMODIC TORTICOLLIS.

(SPASMODIC WRY-NECK.)

A painful tonic or clonic contraction of the muscles of the neck, twisting the head to one side and upwards, or causing marked retraction.

Ætiology.—The patient is of a neurotic type, and often of neurotic extraction. The disease occurs in middle life, and more frequently in women than males. Shock, anxiety, prolonged ill-health, and local injury may precede the symptoms.

The *muscles* affected are (1) the sternomastoid, inclining the head forwards and towards the shoulder of the same side; (2) the splenius of the opposite side, drawing the head backwards and turning the face to its own side; (3) later, the upper part of the trapezii, the trachelo-mastoids, and the other deep neck muscles, on both sides. The *nerves* supplying the sternomastoid and trapezii are the spinal accessory and the anterior primary branches of the second, third, and fourth cervical nerves; those for the splenii and other deep muscles the posterior primary branches of the first five cervical nerves.

No morbid changes, central or peripheral, have been found ; but torticollis is probably due to functional disorder of the cortical centres governing the affected muscles.

Symptoms.—The spasm is either tonic or clonic. Sometimes both forms are combined. In the tonic form the head is retracted and the face turned to one side, the shoulder of that side being also raised. In severe cases the spasm is bilateral. There is then no unilateral rotation, but the head is strongly retracted, and there is wrinkling of the forehead from over-action of the frontalis. In the clonic form there are jerking movements of the muscles, rotating the head from time to time into the position described. The spasm may spread to the facial and brachial muscles. It is aggravated by excitement, and disappears or diminishes in sleep. The affected muscles in time hypertrophy.

Treatment.—Drugs are too often of little use. Bromides and chloral may be of temporary benefit ; morphine is very apt to induce morphinism. Bastian has produced permanent benefit in some cases by keeping the patient asleep for three or four weeks under the influence of chloral and bromide of sodium (10 gr. of each every four hours), wakening him only for food and medicine, which should be given at the same time. Strict isolation must be observed during this treatment. Surgical measures are often necessary. The operation consists in resection of a portion of the spinal accessory nerves and exsection of the posterior branches of the first four or five cervical nerves.

PARALYSIS AGITANS.

Shaking palsy or Parkinson's disease is an affection characterised by rhythmical tremors of certain muscles, progressive weakness, and later a peculiar gait and attitude.

Ætiology.—It affects men more than women, and is rarely found in patients under forty years of age. It is commonest after fifty. The influence of heredity is not marked ; anxiety,

shock, traumatism, are the usual causes assigned, while specific infections appear to have little influence.

Symptoms.—They are very insidious; insomnia, unnatural irritability, and weakness of the limbs generally precede the more characteristic symptoms, which are—

1. *Rhythmical contraction* of certain muscles of the fingers and arms; the fingers are flexed with the thumb resting against the forefinger; the alternating flexion and extension causes movements like rolling pills. The tremors usually spread to the leg of the same side, and finally trunk and face become affected. The movements at first are checked by voluntary effort or support, but later (like chorea) the movements are actually increased on attempting any voluntary restraint. They cease during sleep. The point of distinction between these and other tremors is that in paralysis agitans the tremor is continued during repose, and in whatever position the patient may be, all through the waking hours. The tremor may be confined to one side for several years.

2. *Weakness of the affected muscles.*

3. *Rigidity and contraction*, causing deformity and peculiar gait. The body is bent forwards, the head bowed and held stiffly, and the “vertebra prominens” stands out in bold relief. The arms are flexed at right angles at the elbows, and adducted, while the elbows stand out from the sides. The fingers are flexed at the metacarpo-phalangeal joints, extended at the others. The legs are slightly flexed at hip and knee, and the thighs are slightly adducted. The gait is very characteristic, the first steps are hesitating and slow, but become quick, and the patient appears to trot rather than walk. If the patient be gently pushed forwards or backwards he is unable to stop himself. The movements forward are called “*festination*,” and backwards, retropulsion. There is marked weakness of the muscular actions, but the muscles do not waste, nor are the electrical reactions altered. Actual paralysis occurs only late in the disease. The tendon reflexes may be increased, but there is no ankle-clonus. The rigidity of the facial muscles leads to an expressionless appearance.

4. *Defect of Speech*.—The speech is slow and monotonous, but articulation is not interfered with.

5. *Certain subjective symptoms*, hot flushes, burning pains, formication.

The disease runs a very chronic course, and is fatal usually through some intercurrent complication.

Pathology.—Unknown; probably it is an advanced and premature senile change in the cerebral cortex, rendering certain motor points highly irritable, and diminishing at the same time the inhibitive power of the brain.

Treatment.—Unsatisfactory. Arsenic, sedatives, and attendance to hygiene and diet, avoidance of alcohol, etc., are the chief lines to go upon. Gowers finds cannabis indica combined with arsenic the best drug treatment. Galvanism and passive movements of the limbs may sometimes be of use.

EXOPHTHALMIC GOITRE.

Exophthalmic Goitre is a remarkable condition characterised by—

1. Rapid beating of the heart and palpitation.
2. Enlargement of the thyroid gland.
3. Exophthalmos or projection of the eyeballs, and imperfectly co-ordinated movements of the upper eyelid.
4. Muscular tremor.
5. Certain visceral disturbances.
6. Trophic changes in the epidermal structures.

Morbid Anatomy.—The thyroid is found to be moderately enlarged and exceptionally vascular, the arteries being dilated and tortuous. The epithelium of its vesicles is proliferated, and columnar instead of cubical; the colloid matter is diminished and more mucinoid, and new tubular spaces are formed. The fatty tissues of the orbit are increased in amount. The heart is hypertrophied. Changes in the cervical sympathetic

ganglia and minute hæmorrhages in the brain have been noted.

Pathology.—The pathology of exophthalmic goitre is still a vexed question. Disorders of the sympathetic nervous system, excess or altered composition of the internal secretion of the thyroid, organic or functional disorder of the cerebral nervous system—each of these is regarded as the cause by different authorities. In favour of a thyroid origin are the facts that thyroid extract aggravates the symptoms, that large doses of thyroid have sometimes produced some of them (but not exophthalmos or thyroid hypertrophy), that there is increase of the secreting epithelium, and that partial thyroidectomy affords some measure of relief. It is in favour of a central origin that experimental introduction of large amounts of thyroid gland substance has never caused all the symptoms of the disease; while the mode of onset, often after fright or violent emotion, the much greater frequency (8 or 10 to 1) in women, the history of nervous predisposition so often found, and the nature of the symptoms themselves (tremor, tachycardia, exophthalmos, enlarged thyroid) all point to disturbance of the sympathetic nervous system. It may be impossible as yet to locate the precise seat of the primary disturbance, but certain symptoms, especially those affecting the eyeball (von Graefe's sign, temporary ocular paralysis, etc.), at least suggest a lesion of the medulla. The recurrent laryngeal nerves (branches of the vagus) appear to have a trophic influence upon the thyroid gland, and they contain vasodilator fibres irritation of which increases the blood stream through it. And it is the case that in a few autopsies changes, such as hyperæmia of the medulla and small hæmorrhages in the floor of the fourth ventricle, have been found. The hypertrophy of the gland is often preceded by tachycardia and mental change, and symptoms are often well marked when the hypertrophy is quite slight. Brunton suggests that the disease originates in a central nervous lesion leading to tachycardia and thyroid hypertrophy, which in turn produces auto-

intoxication. Filehne has shown that injury to the upper part of both testiform bodies in rabbits will produce all the characteristic symptoms, but he was not able to produce them all in the same animal.

Ætiology.—The disease is much more frequent in women. It occurs in them most commonly between the ages of fifteen and thirty, in men between thirty and forty-five. The patients are usually of a nervous temperament, and nervous heredity is sometimes apparent. Sudden fright, grief, or shock, are the common exciting causes; depressing diseases or prolonged overstrain may predispose.

Symptoms.—The cardinal symptoms are exophthalmos, thyroid hypertrophy, tachycardia, and muscular tremor. *Tachycardia* and cardiac disturbance appear early and very constantly. The pulse rate varies from 100 to 200 or more per minute. There is anæmia, and from this and the rapid violent action of the heart, dilatation and hypertrophy may follow. Hæmic murmurs and murmurs of relative insufficiency are therefore common. Palpitation is frequent and distressing; the carotids pulsate visibly. *Exophthalmos*, or protrusion of the eyeballs, may be but slight, or so marked that the lids cannot be completely closed. It imparts a staring look of fear or horror to the face. It is sometimes unilateral. It may be caused by (1) venous congestion, or dilatation of the retro-bulbar arteries, (2) overgrowth of the orbital fat, (3) contraction of Müller's muscle. It is attended by certain classical signs—

1. Retraction of the upper lid, so that the sclerotic is seen all round the cornea. *Stellwag's sign*.
2. When the patient looks down, the upper lid follows the movement of the eye slowly and imperfectly, so that a larger area of sclerotic is seen above the cornea. *Von Graefe's sign*.

Other ocular phenomena may occur. There may be ptosis (paresis or paralysis of the branch to the levator palpebræ); imperfect convergence on near vision (*Möbius' sign*);

temporary paresis or paralysis of the third, fourth, or sixth nerve, causing strabismus. The fundus is usually normal. *Thyroid enlargement* is moderate in degree, the right side being usually the larger. It may occur early or late. The gland is soft and elastic at first, later firmer and nodular. Systolic thrill may be felt over it, and systolic murmur heard. *Tremor* may affect the limbs only, or the whole body. It is most notable on holding out the hands, palms downward, and consists of rapid, fine, rhythmic movements of the whole hand, but not the fingers separately. Any one of these symptoms may be absent at a given stage of the disease; of them all, tachycardia is the most constant.

Other symptoms that may occur are digestive disturbances—variable appetite, sometimes vomiting, often diarrhoea; wasting; polyuria, glycosuria, albuminuria; flushing of the head and face, free sweating, occasional pyrexia; cutaneous eruptions; mental alterations—depression, melancholia, or even mania.

Prognosis.—A considerable number of cases recover, more are much benefited by treatment but may relapse, and about 25 per cent ultimately die of complications, diarrhoea, cardiac failure, or acute mania. The duration of a fatal case is from several months to several years.

Treatment.—Physical and mental rest; in severe cases rest in bed. Fresh air; light nutritious diet; avoidance of stimulants; massage; electricity to thyroid (Horsley). Belladonna and bromides are useful sedatives, and digitalis often benefits the heart. Arsenic is often given. Treat special symptoms. In severe cases which are rapidly advancing, operation may be performed. Section of the cervical sympathetic, ligature of the thyroid arteries, section of the isthmus, and partial thyroidectomy have all their advocates. Great benefit, and sometimes cure, has followed operation, but the direct mortality is as high as 12 per cent. Patients should therefore be fully warned of the risk they run.

RAYNAUD'S DISEASE.

A vascular disorder dependent on vasomotor disturbances which cause constriction of the peripheral arterioles, and so induce changes of various degrees conveniently termed—(1) Local syncope; (2) Local asphyxia; (3) Local gangrene. The changes are paroxysmal, and usually symmetrical.

Ætiology.—Practically unknown. The disease is most frequent between the ages of twenty and forty and more frequent in females than in males. Hereditary nervous influence is sometimes apparent, and the patients are usually neurotic. Occasionally some of the specific fevers, malaria, syphilis, functional nervous affections, may be followed by Raynaud's disease. Exposure to cold is the most usual exciting cause.

Pathology.—Unknown. Vasomotor constriction, vasomotor dilatation with subsequent venous engorgement, altered conditions of the blood, etc., have been advanced as the causes. Hyaline changes have been found in the arteries of the cord, and endarteritis obliterans in the arteries of the nerves to the affected limbs. In these nerves a parenchymatous neuritis has also been found. The symmetrical distribution suggests a toxic cause.

Symptoms.—

1. *Local Syncope.*—This condition resembles the dead fingers or toes produced by intense cold—one or more fingers, or the whole hand, may be affected. Often a severe reaction sets in, and the parts numbed before now become intensely red, hot, and painful. The syncope may last from a few minutes to several hours.

2. *Local Asphyxia.*—This condition is characterised by the fingers, toes, ears, and certain patches of skin on the arms and legs becoming intensely congested, livid, and the capillary circulation arrested. The congestion gives rise to swelling, stiffness, and pain; the latter is often succeeded by marked anæsthesia. The duration is similar to that of syncope.

These attacks may occur in winter time for many years as "chilblains."

3. *Local or Symmetrical Gangrene.*—The parts asphyxiated become cold, insensible, and black in colour—*i.e.*, changes identical with necrosis elsewhere. Usually a line of demarcation forms and limits the necrotic condition.

In any given case, these three conditions may occur successively, but more often syncope or asphyxia occurs alone, and either may end in gangrene. Recurrences are frequent, and the paroxysms are markedly periodic.

Complications.—

1. *Paroxysmal Hæmoglobinuria* is frequently present (about 6 per cent of the cases). Other complications are rare, but there may be hæmorrhages, skin eruptions, effusions into serous sacs (joints), delusions, partial coma, or convulsions.

Treatment.—Improve any obvious conditions of ill-health. Locally, make the circulation more brisk by gentle friction, warmth, when it does not increase the pain, as it may sometimes do, and gentle galvanism. Soothe pain by sedatives such as belladonna, morphia, conium, etc.

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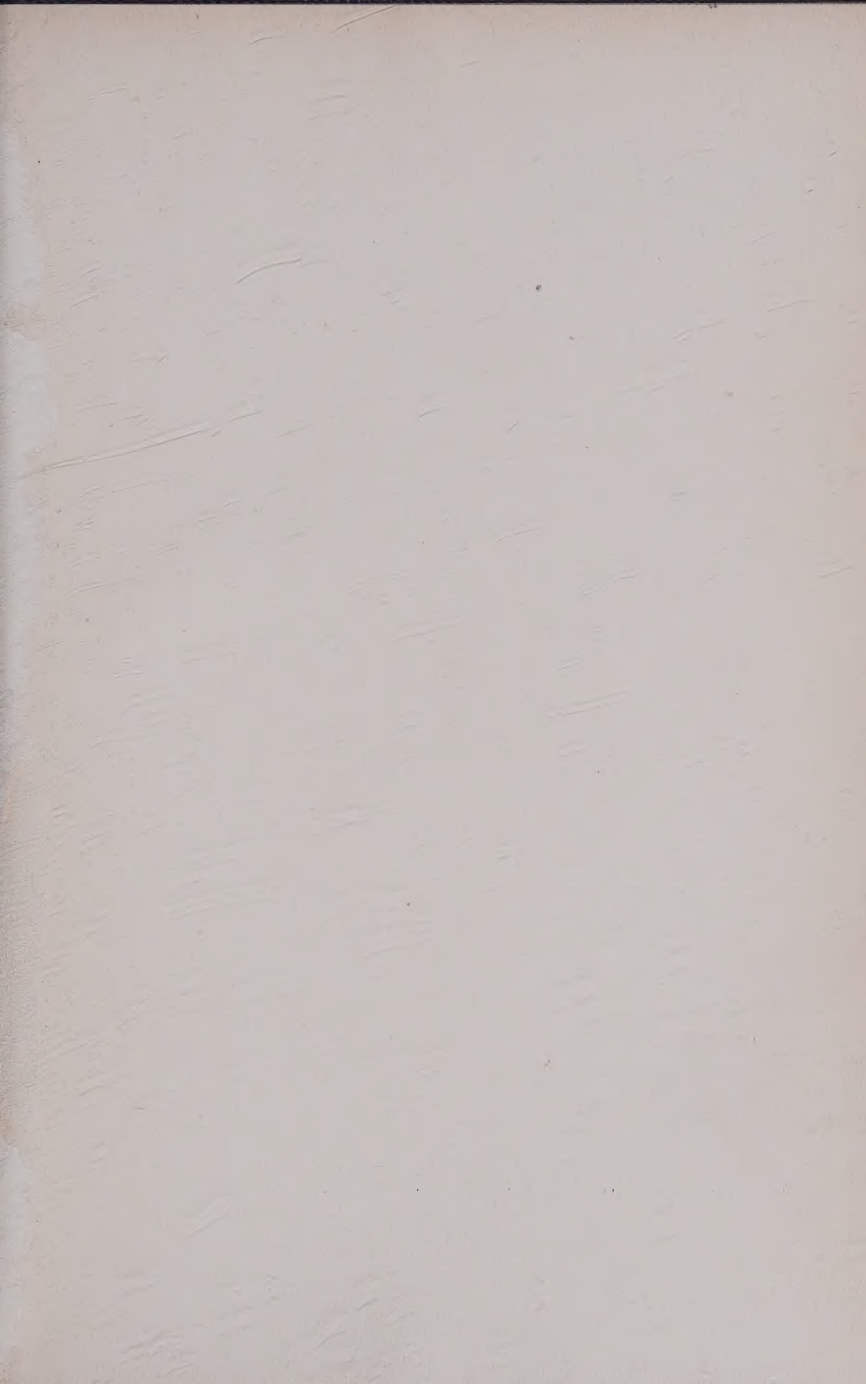
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